Dissertation on

A STUDY ON CLINICAL PROFILE OF UNILATERAL DISC EDEMA

Submitted in partial fulfilment of requirements of

M. S. OPHTHALMOLOGY

BRANCH III

of

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MADRAS MEDICAL COLLEGE

CHENNAI – 600 008

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CHENNAI – 600 032

MAY – 2018

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON CLINICAL PROFILE OF UNILATERAL DISC EDEMA"** is a bonafide record of the research work done by **Dr. ABINAYA. K**, Post graduate in Regional Institute of Ophthalmology, Madras Medical College, Chennai, in partial fulfilment of the regulations laid down by The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2015-2018.

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Last but not the least, my heartful gratitude and sincere thanks to all my patients without whom this endeavour would not have been possible.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled, **"A STUDY ON CLINICAL PROFILE OF UNILATERAL DISC EDEMA"** is a bonafide and genuine research work conducted by me under the guidance of **Prof. Dr. M.V.S. PRAKASH, M.S., D.O.,** Head of the Department, Squint and Neuro Ophthalmology services, Regional Institute of Ophthalmology & Government Ophthalmic Hospital. Chennai – 600 008.

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CERTIFICATE OF APPROVAL

To
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Dear Dr.K.Abinaya,

The Institutional Ethics Committee has considered your request and approved your study titled "**A STUDY ON CLINICAL PROFILE OF UNILATERAL DISC EDEMA" - NO.12012017 (III).**

The following members of Ethics Committee were present in the meeting hold on **24.01.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
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3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
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11.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary - Ethics Committee

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CERTIFICATE

This is to certify that this dissertation work titled **"A STUDY ON CLINICAL PROFILE OF UNILATERAL DISC EDEMA"**" of the candidate **Dr.Abinaya.K ,** with registration Number 221513004 for the award of M.S. in the branch of OPHTHALMOLOGY.

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Guide and Supervisor

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CONTENTS

INTRODUCTION

Disc edema is a common manifestation of variety of disorders. The disc edema can be unilateral or bilateral[1]. Unilateral disc edema can be inflammatory, ischemic, compressive or infiltrative. It may also be an eye opener for detection of certain systemic diseases. Hence, it is very essential for an ophthalmologist to clinically evaluate and differentiate the causes of disc edema.

The presenting signs and symptoms will be different depending upon the cause of the disc edema and the work up for that also should be individualized based on the history and the examination finding. The management and prognosis depends upon the etiology of the disc edema[1]. In most cases the vision can be preserved with appropriate and prompt treatment. If the disc edema is left untreated it can lead to permanent and irreversible blindness due to optic atrophy.

In this study, the clinical profile of each case of unilateral disc edema was analysed in relation to age of presentation, gender, systemic association, risk factor, treatment and prognosis.

REVIEW OF LITERATURE

- Recognition of disc edema as a sign was first done by Hermann Von Helm Holtz by the year 1851 after the invention of the ophthalmoscope.
- In 1860, Albrecht Vongrafe observed 4 patients with brain tumor with swelling of optic nerve head which he called ‘observation staungs papillae’.
- 1908, Parson introduced the term papilledema.
- Paton and Holmes- 1911, differentiated between disc edema due to increased intracranial pressure and optic neuritis

  ▪He described papilledema as a passive edema due to increased intracranial pressure without Primary inflammatory changes and often without disturbance of function,

  Whereas optic neuritis was a swelling of disc associated with inflammation and loss of function
- The Optic Neuritis Treatment Trial (ONTT) is an interventional, multicentric randomised single blind trial and placebo controlled trial[2]. It was started in the year 1988 and finished by the year 2006. This trial is funded by the National Eye Institute.

  The results of the study were

  1) There was no role for oral steroids as it increases the rate of recurrence of optic neuritis.

2) Intravenous methyl prednisolone does hasten the recovery and it does not alter the final visual acuity. This also reduces the risk of developing multiple sclerosis over the next 2 years.
3) With MRI findings the risk of developing multiple sclerosis is 20% whereas without MRI finding the risk is only 3%.
4) Blood investigations and lumbar puncture were not indicated in a typical case of optic neuritis.

- The LONS [the longitudinal optic neuritis study] - it was established as a continuation of ONTT. It was an investigator initiated 15-centre study and is funded by the National Eye Institute.

  The purpose of this study is to assess the benefits and adverse effects of corticosteroids for optic neuritis. It also determines the natural course of vision in patients with optic neuritis and also to identify the risk factors for the development of multiple sclerosis
- The CHAMPS [Controlled High Risk Avonex Multiple Sclerosis Prevention Surveillance] was an interventional [interferon beta 1a], randomised single blind trial and placebo controlled trial

  It analysed the effect of interferon beta 1a treatment in patients with optic neuritis and also with MRI changes suggestive of multiple sclerosis.

It concluded that interferon beta 1a treatment reduces the risk of conversion to multiple sclerosis in high risk patient by 50%

- RP CENTRE STUDY-

  This study was conducted in 2010. This was done to compare the efficacy, visual recovery and side effects of mega dose intravenous methyl prednisolone with intravenous dexamethasone. This study concludes that intravenous dexamethasone was equally effective as intravenous methyl prednislone. Intravenous dexamethasone also had lesser side effect and cost as compared to intravenous methyl prednisolone.

- NATALIZUMAB- it is a newer medication which prevents the immune cell from blood vessel to enter the brain by blocking the receptor called alpha-4- integrin on the surface of white blood cells.

- The IONDT [Ischemic Optic Neuropathy Decompression Trial(1992-1994)][3]

  It is a single-masked, multicenter, randomized clinical trial which analysed the visual outcomes of patients with ischemic optic neuropathy after 24 months of follow-up. It concludes that there is no benefit of optic nerve decompression surgery compared with careful follow-up in patients with non arteritic anterior ischemic optic neuropathy.

DEVELOPMENT OF OPTIC NERVE:

Initially, it was hollow stalk connecting forebrain cavity with optic vesicles. $26^{th}$to 28^{th}day of life, the cavity was lined by neuroectodermal cells[4]. Obliteration of the cavity due to invagination of optic stalk and vesicle at choroidal fissure follows. The fissure closes by 6th week, contains large number of axons surrounding the hyaloid vessels. oligodendroglia and astroglia are differentiation of cells from inner layer of optic stalk. Remnants of hyaloid vessels with glial cells in some individuals may mimic disc edema called bergmeister papillae

OPTIC NERVE HEAD ANATOMY:

Optic nerve is a unique featured nerve as it is surrounded by cerebrospinal fluid and it is the only tract visualized clinically using direct ophthalmoscopy[4]. The ONH [optic nerve head] is formed by axons of ganglion cell layer with 90% contribution from papillomacular bundle, which is the reason for the central and centrocaecal scotoma in optic nerve disorder. It extends from the disc to optic canal, which is located at the sphenoid bone.

The four portions of optic nerve include

- Intra ocular,
- Intra orbital,
- Intracanalicular and

- Intracranial portions.

THE INTRA OCULAR PORTION

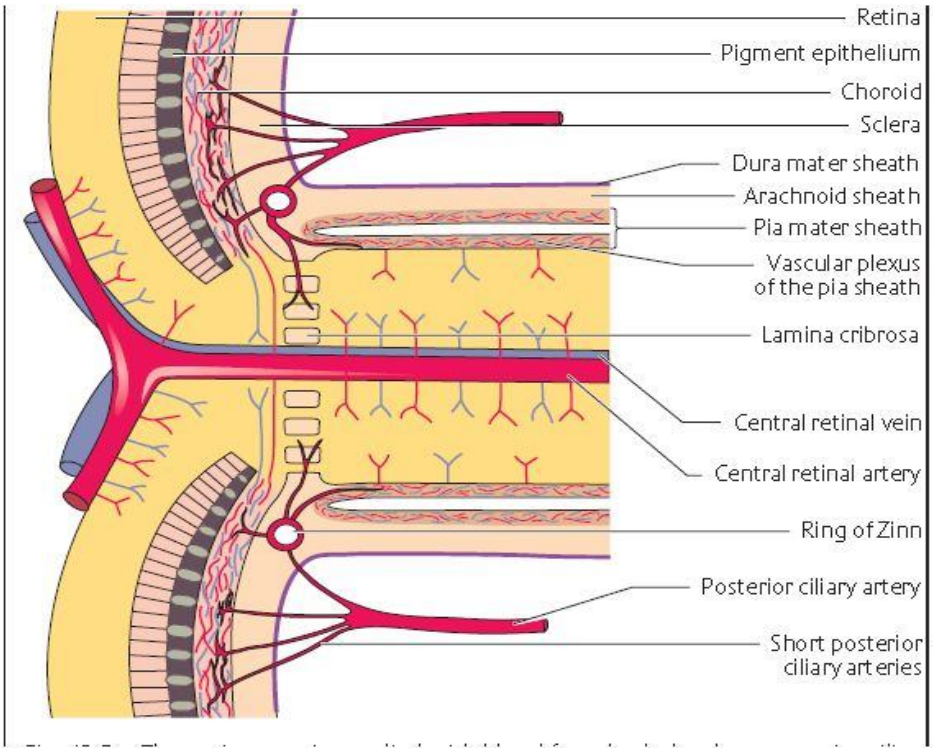
It is further subdivided into retinal, choroidal and scleral portions. The approximate size of optic disc ranges from 1.7 to 2.8mm in diameter. The scleral canal for the optic nerve is 0.5mm long. Posterior to scleral part, the optic nerve becomes myelinated, leading to double of its thickness.

INTRA ORBITAL PORTION:

It courses backward and medially from back of the eye ball to the optic canal in the sphenoid bone and it is covered by all the three layers of meninges. The subarachanoid space around the optic nerve blends with the posterior scleral surface forming a fluid filled ring. The central retinal vein is vulnerable to increased intracranial pressure as it crosses the subarachanoid space. The S shaped bend of this portion prevents it from stretch during intra ocular movements. At its apex, it is surrounded by annulus of Zinn.

INTRA CANALICULAR PORTION

This is the portion of nerve passing through the optic canal, where it is accompanied by sympathetic nerves and ophthalmic artery. The ophthalmic vein passes through the superior orbital fissure.



BLOOD SUPPLY OF OPTIC NERVE

The blood supply is different for different parts of the optic nerve.

1. Blood supply to intraocular part [optic nerve head]

Surface nerve fibre layer

- Capillaries from retinal arterioles –main supply
- Ciliary derived vessel forms cilioretinal artery –occasional supply[5]

Prelaminar region

- Vessels from ciliary region

Lamina cribrosa region

- Short posterior ciliary arteries
- Arterial circle of Zinn

Retrolaminar region

- Ciliary and retinal circulation
- Centripetal branches from pial plexus

2. Blood supply to intraorbital part

Periaxial and axial supply

Periaxial supply

- Ophthalmic artery,long posterior ciliary arteries,lacrimal artery and central artery of retina [before entering the optic nerve]

Axial supply

- Central retinal artery[intra neural branch]
- Central collateral arteries[from central retinal artery]

3. Intra canalicular part

Pial plexus[from ophthalmic artery]

4. Blood supply to intracranial part

Pial plexus

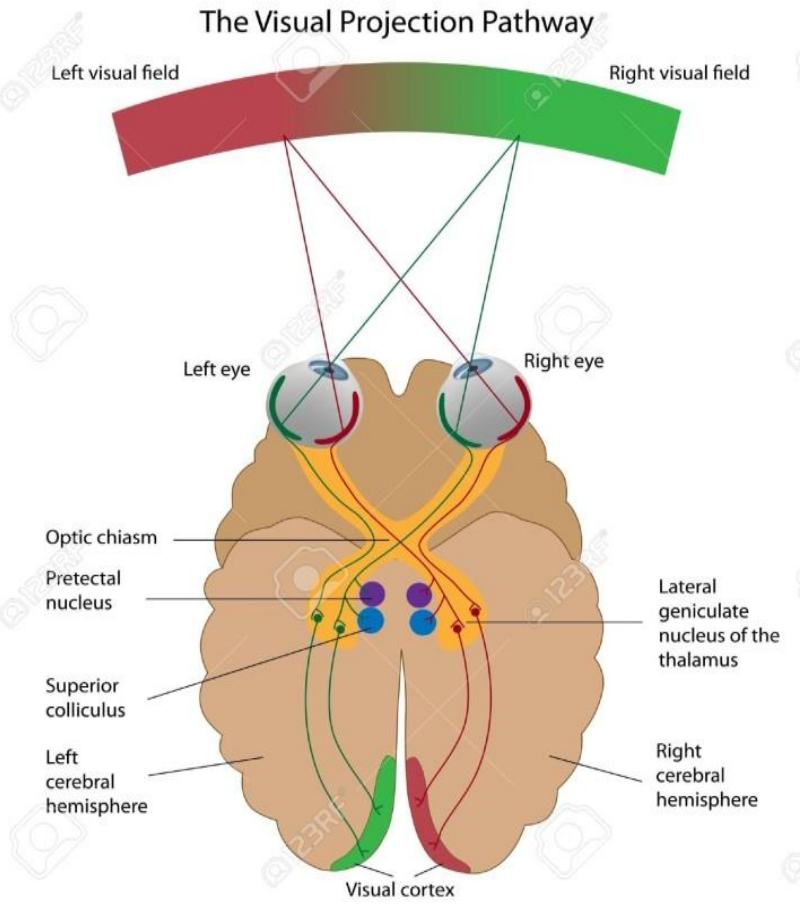
- From direct and indirect branches of internal carotid artery

- From anterior cerebral artery
- From ophthalmic artery
- From anterior communicating artery

VENOUS DRAINAGE OF OPTIC NERVE

- **Optic nerve head**-mainly by central retinal vein[5]
- **Orbital part**-peripheral pial plexus
- **Intracranial part**-pial plexus draining to anterior cerebral and basal vein

VISUAL PATHWAY ANATOMY:



It constitutes

- Retina
- Optic nerve,
- Optic Chiasma,
- Optic tracts,
- Lateral geniculate body (LGB),
- Optic radiation and
- Visual cortex
- Frontal eye field is the other area which is associated with vision.

RETINA:

It is the innermost layer of the eyeball. It consists of inner neurosensory layer and the outer retinal pigment epithelium. It converts the external images into neural impulses and transmits to the brain. Regions of the retina includes

1, **POSTERIOR POLE**

5 to 6mm diameter zone of retina between superior and inferior temporal arteries and is dominated with cone and it consists of single layer of ganglion cells histologically.

2, **MACULA LUTEA**

1.5mm area in the posterior pole consists of xanthophylls, carotenoid pigments which includes lutein and zeaxanthin in the cone axons. This xanthophyll pigment acts as a filter against ultraviolet irradiation.

3, **FOVEA CENTRALIS**

It is the central 0.35mm zone in the Macula. Foveal area is avascular and it depends on the choriocapillaries for its nutrition.

4, **OPTIC DISC**

It is a 1.8mm diameter area located 3mm medial to the centre of fovea. This is the area where all the ganglion cells pierce the sclera to

join the optic nerve. Central retinal vessels emerge from the centre of it and then branches to supply the retina. The central retinal vein lies lateral to the artery.

PERIPHERAL RETINA

It is the part outside the posterior pole. Temporally it measures 23 to 24mm from the disc and nasally it is 18.5mm from the disc. This retina consists of more of rods.

ORA SERRATA

It is the scalloped anterior end of neurosensory retina and is continuing with the columnar non- pigmented cells of pars plana.

RETINAL PIGMENT EPITHELIUM

It is a single layer of neuroectodermal derived cuboidal or columnar epithelium, extends from the optic disc to the ora, where it continues with the pigment epithelium of the pars plana.

It has many vital functions which includes,

- Maintaining the adhesion of neurosensory retina,
- Forming external blood retinal barrier thereby providing selective permeability between the choroid and neurosensory retina,
- Phagocytosis of rods and some extent the contents outer segment,
- Interphotoreceptor matrix synthesis,

- Light absorption thereby providing image resolution by reducing the light scattering,
- Storage of matabolites and vitamin A[4].

The size and shape of RPE varies with age. The hexagonal pattern of arrangement of RPE cells lost as the age advance. As the age advances lipofuscin get deposited in the RPE. RPE varies from eye to eye and it ranges from 4.2 to 6.1 million[4].

NEUROSENSORY RETINA-

It is a thin transparent layer of retina helps in conversion of light stimuli to neural impulses. The photo receptors, bipolar cells and ganglion cells are the three principal neuron cell types involved in relying impulses generated by light. The activity of the above 3 types of cell are modulated by amacrine cells, horizontal cells and other non neuronal elements. The histological section shows eight distinct cell layers which include three layers of nerve cell bodies and two layers of synapses.

The retinal layers include

- RPE,
- Photo Receptors,
- Inner And Outer Segments,
- Outer Nuclear Layer,

- Outer Plexiform Layer,
- Inner Nuclear Layer,
- Inner Plexiform Layer,
- Ganglion Cell Layer And
- The Internal Limiting Membrane.

PHOTO RECEPTORS-

The rods and cones are the two types of photo receptor located in the scleral aspect of the retina. Cones help in fine and spatial resolution and colour vision whereas rods are responsible for contrast sensitivity, brightness and motion. The peripheral retina is rod dominated whereas the cone population increases towards the macula.

The cone and rod cells are slender with an inner and outer segments connected by a stalk (modified cilium). The outer limiting membrane separates the inner and the outer segment from the cell body. The nucleus of photo receptor located in the outer nuclear layer and axons of it passes to the outer plexiform layer where it synapses with the bipolar cells and inter neurones through cone pedicle or rod spherule. The outer segment of rods and cones contains the visual pigment responsible for light absorption and neuro electric impulse generation.

ROD PHOTO RECEPTOR-

It is a 100 to 120 micrometer sized slender cells with the visual pigment rhodopsin in its outer segment which is sensitive to blue green light. These highly sensitive cells are used for vision in dim light. In the outer segment the rhodopsin are contained within membrane bound lamellae which are enclosed within a membrane. These disk or lamellae are synthesized in the basal region of the outer segment and reaches the apex over a course of 10 days where it is enclosed by the apical microvilli of RPE. The outer segments are phagocytosed by the RPE in a circadian fashion. Rods are separated from each other by an interphotoreceptor matrix containing interphotoreceptor binding protein.

The inner part of inner segment is myoid whereas the outer one is called ellipsoid. Ellipsoid part contains numerous mitochondria and myoid region contains numerous organelles indicates the cell is metabolically active.

CONE PHOTO RECEPTOR

There are 3 distinct type of cones exist in humans for short, medium and long wavelength of lights (blue, green and red). Unlike rods the lamellae were not covered by membrane, no circadian phagocytosis of cones outer segment, have greater lifespan than rods and are surrounded by long villous melanin containing apical processes of RPE. The spherules and pedicles are the specialized synaptic

terminals of rods and cones respectively. These contain specialized presynaptic vesicles synapse with bipolar and horizontal cells.

Rod spherule is deeply indented than pedicle. The horizontal cells have deep penetration than bipolar cells which has shallow penetration. The cone pedicles are broader and pyramidal in shape with 12 indentations, each of which contains 3 neuronal triads. The central process of triad is for midget bipolar cells and the peripheral processes for horizontal cells.

BIPOLAR CELLS

They transmit impulses from photo receptors to the ganglion cells. Their cell processes are located in the inner nuclear layer and oriented radially. In the central retinal area the ratio of cone, bipolar cell and ganglion cell is 1:1:1, whereas in the retinal periphery one bipolar cell receives input from 50 to 100 rods.

GANGLION CELL

Ganglion cell layer located between the inner plexiform layer and nerve fibre layer. The axons from it synapse with the lateral geniculate body. The axons are separated and ensheathed by glial cells. Behind the lamina cribrosa, the axons get myelinated by oligodendrocytes. There are several layers of ganglion cell in the central retina and only one in the peripheral retina.

RETINAL NEURO GLIA

It includes astrocytes, muller cells, microglial cells. Astrocytes are predominantly located within the vitreal aspect of inner nuclear layer and form an irregular scaffold between the vessels and neurones perpendicular to the muller cells. It helps in isolation of receptive surface of neurones and contains abundant cytoplasmic structural fibrils consisting of glial fibrillary acid protein.

Muller cells are the main cells for supporting glial cells of retina. It extends from internal limiting membrane to external limiting membrane. It helps in intracellular transport and secretion and help to nourish and maintain outer retina[4].

Microglial cells are the highly specialized cells of mononuclear phagocytic system. In retina, they are located in three strata. It helps in tissue homeostasis and host defense. On injury to the retina, they become activated.

OPTIC NERVE

Intracranial portion of the optic nerve extends from optic canal to optic Chiasma at the floor of 3rd ventricle. It relations in that part includes the olfactory nerve, frontal lobe and the anterior cerebral artery. The internal carotid artery lie lateral to chiasma, as it emerges from the roof of cavernous sinus.

OPTIC CHIASMA

It is a 12 x 8mm flattened quadrangular area located at the junction of anterior wall and the Floor of 3rd ventricle and 5 to 10mm above the Sella diaphragmatica. The infundibulum lies behind and below the Chiasm and between the mamillary bodies. The anterior communicating artery lies above the optic Chiasma. The optic nerve fibres from the nasal hemiretina cross at the chiasm. This crossing is essential for binocular single vision. But before crossing, it might take a short loop in the ipsilateral optic tract or the contralateral optic nerve.

OPTIC TRACTS

It extends from the optic chiasma to the lateral geniculate body mostly and some 10% of the optic tract fibres to the superior colliculus and the pretectum. Temporally tracts wind around the cerebral peduncles and divide into a large lateral branch and a small medial root. The medial root consists of 6 groups of fibres, the 3 of which is connected to superior colliculus which is involved in visual association pathways, visual grasp reflex, automatic scanning of images. The other three to either the parvocelullar reticular formation involved in arousal function or to the pretectal nucleus involved in the pupillary light reflex or to the retinohypothalamic tract.

The large lateral root goes backwards and laterally to join the lateral geniculate body (part of thalamus). It is attached to the outer wall

of third ventricle through a narrow band of tissue medially. It goes around the cerebral peduncles by passing above the dorsum sella and crosses third nerve from medial to lateral. Posterior cerebral artery runs bellow the optic tracts and lies parallel to it[4].

LATERAL GENICULATE BODY [LGB]

It is an ovoid projection located in the posteroinferior aspect of the thalamus. The parts of LGB include head, body, spur and hilum. It consists of six laminae or cell layers where the tract fibres terminate. The coronal section of it shows the layers of cell nuclei separated by white matter. The nasal part of contralateral eye terminates in cell bodies located in layers 1, 4, 6, where as the fibres from temporal retina of ipsilateral eye terminate on layers 2, 3, 5.

The fibres from the lower quadrant of the retina terminate on the lateral part of LGB and that of upper quadrant on its medial aspect. The Macula fibres terminate on the large central wedge area of LGB. The most of LGB fibres passes to the visual cortex (area 17) through optic radiations. LGB also has its input from areas 17, 18, 19, occulomotor centre and reticular formation.

GENICULOCALCARINE TRACT/ OPTIC RADIATION

This tract starts from the cell body located in LGB to the occipital cortex (visual striate cortex). These fibres form a loop called Meyers

loop which is a forward and inferiorly directed fan shaped loop. It then reaches the temporal lobe by passing around the inferior horn of lateral ventricle and then turns medially to join the occipital cortex by passing along the posterior horn of lateral ventricle. The farthest fibres that swing into the loop are from the peripheral retina.

PRIMARY VISUAL CORTEX

The fibres from the optic radiation enter the primary visual cortex area 17, which lies deep in the calcarine sulcus. The primary visual cortex extends above and below the margin of the calcarine sulcus on the medial surface of occipital cortex. It extends posteriorly up to the pole of occipital lobe and anteriorly up to the parieto-occipital sulcus. The cuneus gyrus and the lingual gyrus are the areas which located above and below the calcarine sulcus respectively. The upper lip of calcarine sulcus contains fibres from the superior retinal quadrants. The Macula fibres represent one- third of the visual cortex. The stria of Gennari is a conspicuous area where the myelinated fibre of geniculocalcarine tract enters the cortex, the layer 4 of the cortex.

The predominant cell type is a small stellate cell unlike the pyramidal cells in other cortical areas. The ocular dominance column of this “layer 4” receives input from both right and the left eye and the matching points of retina from both eyes are arranged side by side in contiguous columns. The cells of laminae two and three gives output to

secondary visual cortex and lamina five to the superior colliculus and lamina six feed forward to the lateral geniculate nucleus.

SECONDARY VISUAL CORTEX VISUAL ASSOCIATION AREAS

These are area number 18 and 19 located above and below the area 17. It extends over the lateral surface of occipital cortex. This also contains the six layers, but less extensive and it receives input from the thalamus, the area 17, pulvinar and other cortical areas. The outputs are to the area number 7 involved in steropsis and movement, area 37 involved in recognition of faces, frontal eye field and occulomotor nerve involved in sensory motor eye coordination. This also helps in integration of information from two halves of the visual field through the commissural fibres in the splenium of the corpus callosum.

FRONTAL EYE FIELD

It is the broadmann area number 6, 8, 9, involved in voluntary eye movement, (saccade). The fibres from the frontal eye field passes to the superior colliculus and to the 3rd, 4th and 6th cranial nerve nucleus and the anterior horn cells in the cervical part of spinal cord, thereby helping in co-ordination of head and neck movement with the eye movement[4].

OVERVIEW OF OPTIC DISC PATHOLOGY

Eighty percent of the optic nerve fibres are from the macula, hence the disease of macula and optic nerve mimic each other[6]. Microscopically the optic nerve possesses the oligodendrocytes for Schwann cell, microglia for macrophages and astrocytes for fibroblasts[6]. The unique feature of optic nerve fibre is that they do not have the power to regenerate once damaged. The optic nerve can be affected in various conditions.

The optic disc edema in any optic nerve disorder is due to

- Accumulation of fluid around the optic disc
- Ischemia by compromising the blood supply
- Inflammation around the nerve
- Direct compression or toxic effects of alcohol or drugs.
- Direct or indirect trauma to the optic nerve.
- Congenital anomalies due to abnormal embryogenesis.

Optic nerve disorders can be detected by checking visual acuity, colour vision, pupillary assessment, field charting and by ophthalmoscopic examination of the optic nerve head. Early detection can be possible by checking contrast sensitivity and stereoacuity. The retinal diseases can be differentiated from the optic nerve disorders by checking pupillary reflex and the photo stress test. In retinal disorders

the pupillary light reflex will be normal and photo stress test will be positive[6].

The optic nerve disorder produces variety of field defects. But it can be broadly classified into central, centrocaecal, never fibre bundle defects. The best method to detect the visual field defect is by Humphrey static automated perimetry using 30-2 programme for central field or by neurological field.

The optic disc in any pathology may appear as an edematous disc, a normal disc, hyperaemic disc or a pale disc[6]. Any disc pathology or optic neuropathy may finally lead to optic atrophy.

PATHOLOGY OF OPTIC DISC SWELLING

Optic disc swelling is due to arrest or obstruction of axoplasmic flow at the lamina cribrosa. It may be due to various pathological conditions like ischemia, infiltration, inflammation, compression, metabolic and toxic damage[7, 8].

Ophthalmoloscopically, the early disc edema usually presents as superior and inferior margin blur and the increasing swelling can obscure the blood vessels at the disc margin. Hyperemic disc with absent spontaneous venous pulsation may be seen [Spontaneous venous pulsation is normally absent in 20% of the people]. In the stage of fully developed disc edema, intra retinal hemorrhages, infarcts leading to soft exudate and hard exudates may be seen.

In the long course, after several months, the hemorrhages and hard exudates may resolve and the hyperemia is replaced by milky gray appearance due to gliosis[7]. The presence of optociliary shunt, neo vascular membrane with subretinal hemorrhages and serous fluid are also not uncommon. The final fate of any optic nerve disease is atrophy. The type of atrophy in any disc edema is secondary with dirty yellow colour disc with ill defined disc margin with surrounding vascular sheathing. Once the atrophy develops, the optic nerve does not swell[7].

EVALUATION OF UNILATERAL DISC EDEMA

HISTORY

- Unilateral or bilateral involvement

In Unilateral involvement→ lesion is before the optic chiasm

In Bilateral involvement→ binocular involvement, chiasmal or retrochiasmal lesion[9]

- **ONSET OF VISION LOSS**

  Sudden onset-ischemia [painless] or optic neuritis [painful eye movements]

  Gradual onset [over days to weeks] → Inflammatory optic neuropathy

  Gradual onset [over months to years] → Compresssive optic neuropathy[9]

- **ASSOCIATED SYMPTOMS**

  Periorbital pain→ Optic Neuritis

  Diplopia, hemiparesis→Demyelination

  Jaw claudication and headache→Arteritic aion

OCULAR EXAMINATION

BEST CORRECTED VISUAL ACUITY

COLOUR VISION using pseudo isochromatic colour plates

Other standard tests for colour vision detection are Farnsworth panel D-15 test, Farnsworth Munsell 100 hue test.

PUPILLARY ASSESSMENT

Light directed to one pupil causes constriction of other pupil and the response should be equal. If there is difference in pupillary response from one eye to the other eye, then it is called as relative afffernt papillary defect [RAPD]. It can be demonstrated using swinging flash light test. If one pupil does not act due to trauma or synechiae the

consensual response should be seen in the other eye. RAPD can be graded from 1 to 4 depending upon the severity and can be quantified using neutral density filter[9].

The strength of the neutral density filter which is needed in the normal eye to reach the balance point is the measure of RAPD. A mild contralateral RAPD can be seen in optic tract lesion[9].

FUNDUS EXAMINATION

The fundus examination using direct ophthalmoscopy or slit lamp biomicroscopy using +78D or +90D lens, one can assess the optic nerve head pathology

Pathology	Fundus features
Optic neuritis	Disc edema with blurred margin and hyperemia
AION	Disc edema with either diffuse or sectoral pallid edema with splinter haemorrhages and venous dilatation
Inflammatory disc edema[posterior uveitis]	Disc edema associated with vitritis and sometimes with anterior chamber reaction
Neuroretinitis	Disc edema associated with macular hard exudates arranged in a star pattern
Compressive optic neuropathy	Disc edema associated with choroidal folds and absent SVP[spontaneous venous pulsation]

In any type of optic nerve pathology the disc will go for atrophy within 4 to 6 weeks and the type of optic atrophy will be secondary.

Secondary optic atrophy

In secondary optic atrophy, the disc is dirty yellow in colour with ill defined margin and the surrounding arteries show attenuation and sheathing. The pathology in this type of optic atrophy is nerve fibre degeneration with excessive proliferation of glial tissue.

Visual field evaluation-

- Standard method is to evaluate using automated perimetry.
- Most of the optic nerve head pathology shows central or centrocaecal scotoma.
- In AION, the most common field defect is altitudinal field defect.

CAUSES OF UNILATERAL DISC EDEMA:

TRUE DISC SWELLING

1. Optic neuritis or papillitis of optic nerve head (this can lead to sudden loss of vision with subsequent improvement)
2. Anterior ischaemic optic neuropathy (this will lead to sudden vision loss with no improvement usually)
3. Compressive lesions due to orbital tumors like haemangioma, glioma etc (this causes slow progressive loss of vision)

4. Papillophlebitis or disseminated vasculitis or any posterior uveitis (inflammatory)
5. Central retinal vein occlusion
6. Infiltrative disorders such as leukemia, lymphoma etc
7. Ocular hypotony
8. Foster Kennedy syndrome(true papilledema in one eye with optic atrophy in the other eye, is mainly due to frontal lobe tumors like aesthesioneuroblastoma)
9. Pseudo foster Kennedy syndrome[6,7]

DISC ELEVATION WITHOUT TRUE SWELLING

1. Optic disc anomalies [10]
2. Optic disc Drusen
3. Optic disc myelinated nerve fibres
4. Tilted and crowded disc[7]

OPTIC NEURITIS

It is the inflammation of the optic nerve. Anywhere along the course the optic nerve might get involved. It is classified into two main categories

1. Affection of the optic nerve that can be seen through opthalmoscope

This includes

- papillitis and
- neuro retinitis

1. If the optic nerve is affected proximal to the intra ocular portion, it does not show any optic nerve head changes ophthalmoscopically. It is called as Retrobulbar neuritis [6]

ETIOLOGY OF OPTIC NEURITIS:

1. Idiopathic

2. Demyelinating diseases

- Isolated
- Associated with multiple sclerosis
- neuromyelitis optica(Devics disease)
- Myelinoclastic diffuse sclerosis (schilder's disease or encephalitis periaxialis diffusa)
- Encephalitis periaxialis concentrica (concentric sclerosis of balo)

3. Infectious and para infectious optic neuritis

- LOCAL

  Endophthalmitis , orbital cellulitis, sinusitis, spread from meninges, brain, base of skull

- SYSTEMIC
  - ✓ Viral (mainly measles, mumps, chicken pox and sometimes even follows vaccination)
  - ✓ Bacterial (syphilis, tuberculosis, cat scratch disease)
  - ✓ Fungal (cryptococcal, histoplasmosis)
  - ✓ Protozoval (toxoplasmosis, toxocariasis)
  - ✓ Parasitic (Cysticercosis)

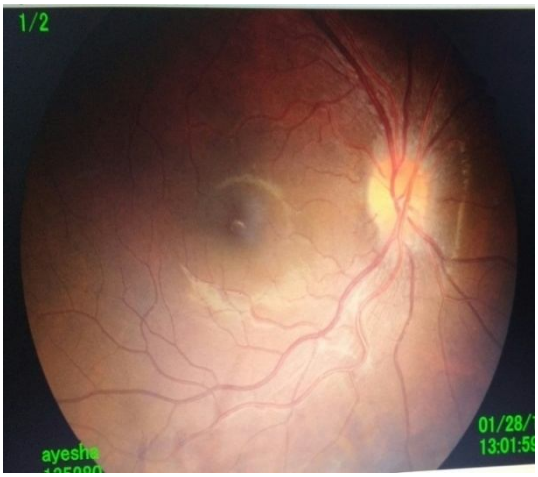
4. Immune mediated

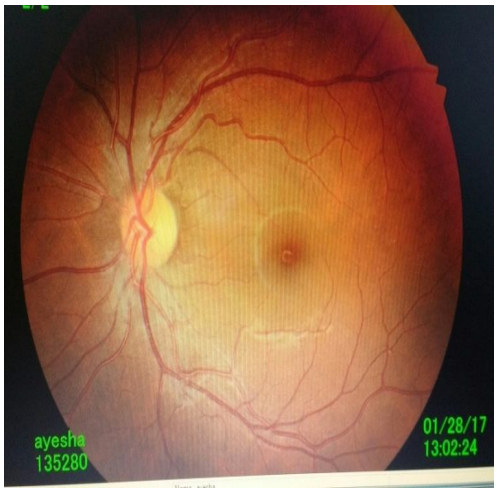
- Local- posterior uveitis, sympathetic ophthalmitis
- Systemic – sarcoidosis , wegener's granulomatosis, acute disseminated encephalomyelitis

5. metabolic disorder

- Diabetes
- Anaemia
- Avitaminosis[7]
- Starvation.

IDIOPATHIC DEMYELINATING OPTIC NEURITIS





It is a primary demyelinating process which occurs in isolation or in patients with multiple sclerosis. Even in isolated cases, there is a higher risk of developing multiple sclerosis than in normal population. It can occur in any of the three forms →acute, chronic and subclinical forms.

Acute Demyelinating optic neuritis is the most common form[11]. Females are commonly affected with a female male ratio of 3:1 and majority of patients are between the ages of 20 to 50 years. The two most common symptoms are defective vision and pain. The degree of vision loss varies from mild reduction to complete loss of vision with no perception of light. The pain is very severe, may precede the onset of vision loss or occurs concurrently.

The contrast sensitivity and colour vision are impaired in most of the cases with subtle changes in colour which can be checked using Farnsworth-Munsell 100-hues test. The loss of visual field can be mild to severe with any type of field defect. In ONTT, the typical central or centrocaecal defect occurs in only 8% of the patient. In swinging flash light test, the relative afferent pupillary defect is demonstrated in all cases of unilateral optic neuritis and it is absent in bilateral optic neuropathy.

About one third of optic neuritis cases present with disc edema. The severity of vision loss does not correlate with the degree of disc edema. The peripapillary disc hemorrhages and sectoral edema are less common in optic neuritis compared to ischaemic optic neuropathy.

In some cases of optic neuritis minimal anterior or posterior uveitis may present. In extensive cellular reaction, one should suspect other inflammatory or infectious causes of optic neuritis like sarcoidosis,

syphilis, lymes disease or cat scratch disease. Symptomatic bilateral disease is most common in children than in adults. In such bilateral cases infectious causes should be ruled out. Even with improvement in visual acuity and colour vision, the pallor settles in approximately 4 to 8 weeks of onset[7].

VISUAL PROGNOSIS:

This acute demyelinating optic neuritis worsens over days to 2 weeks and then improves. Initially the improvement is rapid and then this improvement levels off and continues till 1 year. Less than 10% of patients have permanent loss of vision of less than 20/40. The colour vision and the field defect improve in conjunction with visual acuity. Even though there is a good visual improvement, some patients might complain of movement induced photopsia. The recurrence of optic neuritis may occur in either eye. The recurrence of optic neuritis is less with intravenous steroids compared to oral prednisone (ONTT). The visual recovery is good in optic neuritis[12].

RISK OF MULTIPLE SCLEROSIS

Optic neuritis is a presenting feature in 20% of multiple sclerosis patients and 50% of multiple sclerosis patients develop optic neuritis[7]

INVESTIGATIONS:

When to do investigation?

To rule out compressive optic neuropathy

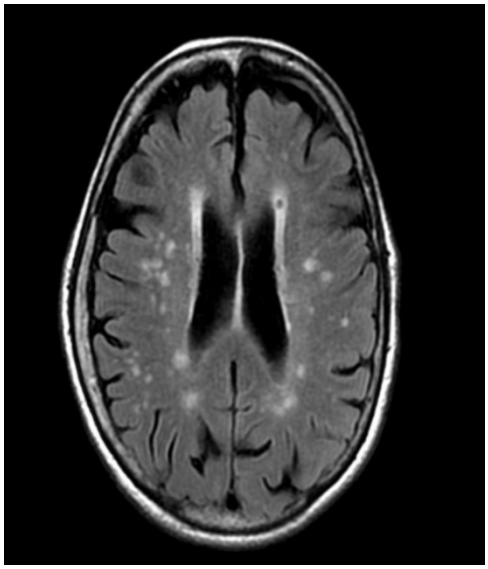
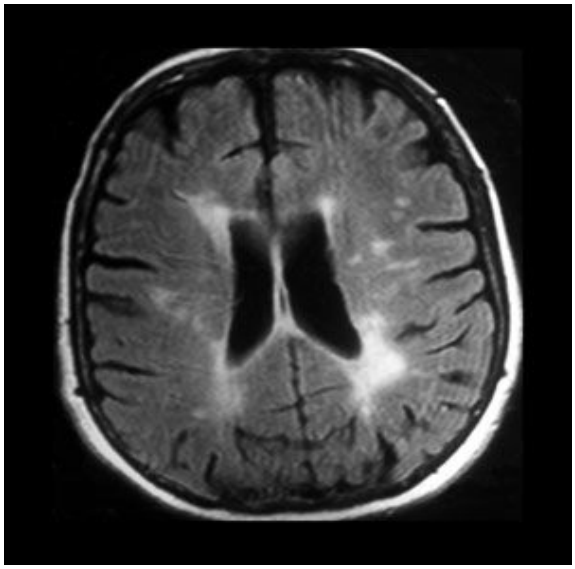
To rule out causes of inflammation other than demyelination

To determine the visual prognosis and the neurological prognosis of optic neuritis.

MRI Brain-

It can detect demyelinating lesion of optic nerve (foci of T2 hyperintense area of enhancement), enlargement of optic nerve. But it is nonspecific and can also be seen in other inflammatory and infectious causes of optic neuritis.

MRI Brain also acts as a strong predictor of multiple sclerosis in patients with acute idiopathic isolated optic neuritis[7]. Two or more brain lesions in the form of signal abnormalities in the white matter of brain, usually in the periventricular region is seen in 27 to 60% of isolated optic neuritis patient.

BLOOD INVESTIGATION-

- ✓ Total WBC count, differential count, ESR.
- ✓ Other blood investigation like
    - ACE levels to rule out sarcoidosis,
    - ANA levels to rule out SLE
- ✓ Investigation to find the infectious causes of optic neuritis like
    - Lymes disease,
    - TB [, Mantoux test, Chest X-ray],
    - Syphilis [VDRL],
    - Cat scratch disease

These investigations are tailored for each patient depending upon the symptoms and clinical signs.

CSF analysis for oligoclonal bands is not a must to rule out multiple sclerosis, but it helps in diagnosing other infective and inflammatory causes of optic neuritis[7].

VEP (visually evoked potential)

Prolongation of latency in VEP is seen in optic nerve disorders.

TREATMENT

The treatment is given as per optic neuritis treatment trial [ONTT] for the acute idiopathic optic neuritis. It is intravenous methyl prednisolone 250 mg QID for 3 days followed by oral prednisolone 1 mg/kg for 11 days. The visual recovery is fast in this treatment and it may delay the onset of multiple sclerosis but it does not change the long term prognosis. The intravenous methyl prednisolone reduces the rate of development of multiple sclerosis compared to oral prednisolone alone during the first two years. The CHAMPS study [Controlled High MS Risk Avones MS Prevention Study] and ETOMS [The Early Treatment of Multiple Sclerosis Study] shows the reduction in the three year risk of developing multiple sclerosis in a group of patients receiving interferon –beta-1a[13].

CHRONIC DEMYELINATING OPTIC NEURITIS

This less common type shows slow progressive loss of vision in one or both eyes. This type of optic neuritis usually develops after the onset of signs and symptoms of multiple sclerosis.

SUBCLINICAL OPTIC NEURITIS

This type of optic neuritis does not have any visual symptom but has a subtle disturbance of colour vision which can be found out in autopsy studies of patient with multiple sclerosis.

OTHER DEMYELINATING DISEASES

It includes

- Neuromyelitis Optica [Devics disease]
- Myelinoclastic Diffuse Sclerosis [Schilders disease]
- Encephalitis periaxialis Concentrica [Concentric sclerosis of balo]

NEUROMYELITIS OPTICA

It is a demyelinating disease of spinal cord and optic nerve causing acute or subacute loss of vision in one or both eyes associated with paraplegia or paraparesis. The acute optic neuropathy can be preceded or followed by longitudinally extensive transverse myelitis [LETM] within days to weeks. Sometimes it may take years to develop. No sex delineation is seen and it is a disease of young adults. The vision loss is rapid and severe compared to multiple sclerosis optic neuritis and the visual recovery is also poor. The visual recovery usually begins within 1 to 2 weeks and the maximum improvement takes several months which is also incomplete.

Diagnosis of NMO can be made out with MRI Spine showing Longitudinally Extensive Transverse Myelitis [LETM] and with a serology positive for NMO-IgG antibodies. There is no specific treatment for NMO but the patient can be given IV methyl prednisolone 500 mg BD for 5 days followed by low dose steroids with immunosuppressives like azathioprine for longer duration.

SCHILDERS DISEASE

It is a non inherited demyelinating disease with severe, selective myelinoclasia affects the entire cerebral hemisphere extends across the corpus callosum, brainstem, cerebellum, spinal cord. The histopathology is similar to multiple sclerosis which includes fibrillary gliosis, swollen astrocytes, perivascular cuffing and is considered as a variant of MS. It causes cortical blindness, central deafness, varying degree of hemiparesis, nystagmus, intention tremor etc. The prognosis is very poor with progressive unremitting course which ends in death. Corticosteroids and immunosuppressants can be tried.

CONCENTRIC SCLEROSIS OF BALO

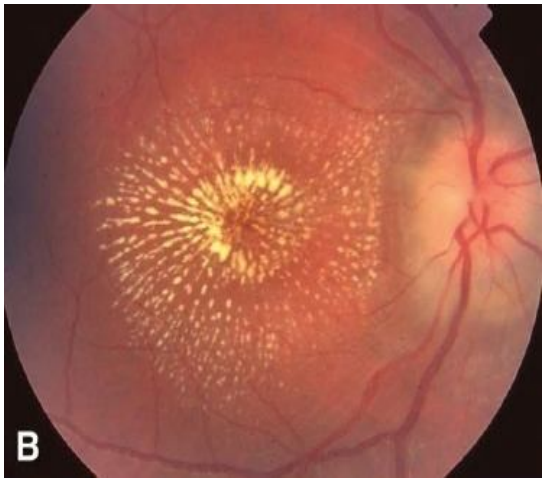
It is a condition clinically similar to Schilders disease but pathologically different. The pathology shows alternating bands of demyelination and remyelination in the cerebral white matter causing concentric bands. Prompt treatment with systemic corticosteroids shows good results and thus early diagnosis is mandatory.

NEURORETINITIS

It causes acute unilateral loss of vision with optic disc swelling and star pattern of hard exudates around the fovea. The causes can be divided into infectious and non infectious. If no cause is found out it is called as Leber's Idiopathic Stellate Neuroretinitis[15]. The infectious causes include cat-scratch disesase [Bartonella henselae], syphilis

[secondary and tertiary syphilis], lymes disease, leptospirosis and viral causes include herpes simplex, hepatitis B, mumps etc[14]. Toxoplasmosis, toxocariasis and histoplasmosis are the other presumed causes.

Sarcoidosis is an important non infectious inflammatory cause of neuroretinitis. It usually affects person of 3rd and 4th decade with no sex predilection. Posterior inflammatory signs like vitritis, venous sheathing and anterior chamber reaction may present. It is usually self limited with good visual prognosis. The optic disc edema resolves over a period of 6 to 8 weeks with no evidence of disc pallor and the hard exudates takes 6 to 12 months to resolve. There is no risk for the development of MS in future. The treatment depends upon the underlying cause and the prognosis is usually good.



INFECTIOUS AND PARAINFECTIOUS CAUSE OF OPTIC NEURITIS

It is a unilateral or bilateral optic neuritis common in children, typically follows viral or less often bacterial or post vaccination by 1 to 3 weeks. It is an immunological process producing demyelination of optic nerve. Most of the patient does not have any neurological dysfunction, but sometimes the patient present with meningitis, meningoencephalitis as in acute demyelinating meningoencephalitis[ADEM]. The visual recovery is usually excellent without treatment but corticosteroids can be given for the fast recovery.

The infectious viruses are CMV, HAV[hepatitis A virus], HIV, EBV, Measles, Mumps, Chicken pox and the bacterial causes are Syphilis, lymes disease ,cat-scratch disease, TB , Typhoid fever, meningococcal infection etc. Spread of infection from the sphenoid sinus can produce isolated loss of vision. Aspergillosis and other fungal infections like mucormycosis in diabetic patients can also cause optic neuritis.

INFLAMMATORY OPTIC NEURITIS

Sarcoidosis is an important cause for inflammatory optic neuritis. Clinically optic disc may have a lumpy white appearance with vitritis and anterior chamber reaction may present. It is extremely sensitive

sensitive to steroids and rapid recovery is noted. Other causes are SLE [1%], PAN [polyarteritis nodosa] and other vasculitides.

OPTIC PERINEURITIS

It is a perioptic neuritis involves the periphery of the optic nerve. It is a form of orbital inflammatory pseudo tumour, come in isolation or associated with scleritis and myositis and is not associated with MS. If it bilateral differentiation from papilloedema is essential. As there is no optic nerve involvement, there is no visual dysfunction.

ISCHEMIC OPTIC NEUROPATHIES

- ANTERIOR
- POSTERIOR

ANTERIOR ISCHEMIC OPTIC NEUROPATHY

It is divided into arteritic [GCA] and non arteritic.

NON ARTERITIC AION

The most cases of AION are non arteritic type. The patient presents with painless loss of vision over hours to days. The mean age of onset ranges from 55 to 65 years with no gender predilection. It usually produces less severe vision loss {>6/60} and the colour vision loss tends to parallel with vision loss. The optic disc edema may be diffuse or segmental hyperaemia, but pallor is less common compared to arteritic form. The visual field defect follows the pattern of optic nerve damage and the most common form is inferior altitudinal loss.

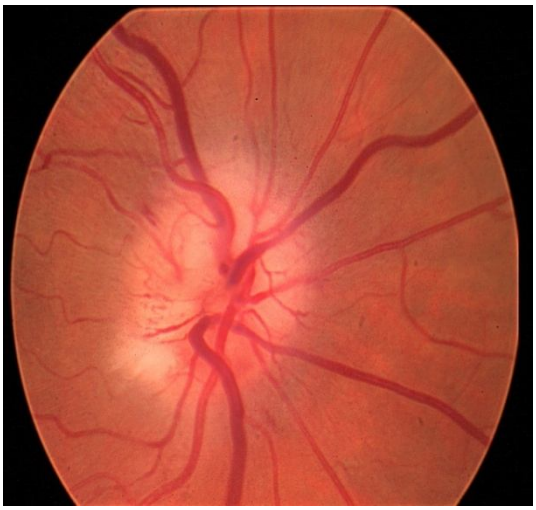
Peripapillary retinal haemorrhages are common and the retinal arteries are focally narrowed in that region. The optic disc in the contralateral eye shows absent or small cup. The para optic branches of short posterior ciliary arteries play the major role in NAION. In the setting of carotid occlusive disease, general ocular ischemia is more common compared to AION. So majority cases of NAION are unrelated to carotid disease. The mechanism in the development of AION remains unclear. The proposed mechanisms are local arteriosclerosis with or without thrombosis, embolisation from the remote source, generalized hypo perfusion, failure of auto regulation.

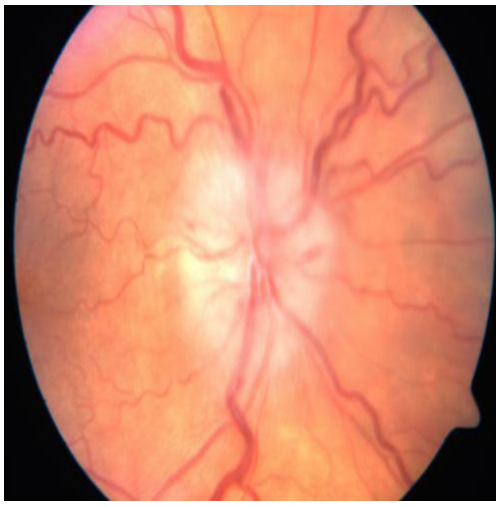
The mechanism in crowded disc causing ischemia is similar to a compartment syndrome[16,17]. The other risk factors for AION are nocturnal hypotension, sleep apnoea, vasculopathy and coagulopathy. It is also seen in conditions where optic nerve head perfusion is affected due to micro vascular occlusion in diseases like hypertension and diabetes mellitus. There is an incomplete study on risk of stroke, myocardial infarction and death in patients with NAION in future. The optic disc structure or other features which compromise optic nerve head perfusion and there by ischemia are optic nerve head drusen, elevated intraocular pressure, cataract surgery and migraine. The drugs implicated in the development of NAION are sildenafil and interferon alpha.

Most cases of NAION show no significant visual improvement and the vision loss stabilises within 2 months. After stabilisation of vision, deterioration is very rare. The disc goes for secondary optic atrophy within 6 to 8 weeks. The occurrence in second eye is less common compared to arteritic AION. The second eye occurrence gives a picture of pseudo- Foster Kennedy syndrome.

The patients under the age of 50 without any systemic illness and without any crowded disc in other eye should undergo investigations for coagulopathy, vasculopathy and the NAION must be differentiated from idiopathic optic neuritis, syphilitic and sarcoid related optic neuritis. Neuroimaging is indicated if it is associated with severe pain and the disease follows an atypical course like persisting disc edema or progressing course or recurrence after 2 months.

There is no proven treatment or prophylactic measures for NAION. According to IONDT [ischemic optic neuropathy decompression trial], optic nerve decompression in fact worsens neuropathy in NAION.





Difference between arteritic and non arteritic AION

	ARTERITIC	**NON ARTERITIC**
AGE	>70	>50
SEX	F>M	No gender predilection
VISUAL ACUITY	<6/60	>6/60
ASSOCIATED SYMPTOMS	Present	Absent
ESR	Raised	Normal
CONTRALATERAL INVOLVEMENT	55 to 85%	15 To45%
TREATMENT	Steroids	Nothing proven

POSTERIOR ISCHEMIC OPTIC NEUROPATHY

It is due to ischemia of retro bulbar part of optic nerve which has a different blood supply compared to the anterior part of optic nerve which is supplied by posterior ciliary arteries. This retrobulbar part is supplied by pial plexus, anterior cerebral artery etc. The causes for PION include GCA, other causes of arteritis, causes of NAION, hypotension, severe blood loss.

Most of the time the optic disc is normal, but sometimes it can present as disc edema due to migration of fluid anteriorly from the ischemic area.

RADIATION OPTIC NEUROPATHY

It is due to irradiation to brain tumours or paranasal sinus lesion or an orbital lesion. It presents mostly as pale disc. Sometimes due to associated retinal involvement disc edema may present. The onset of symptoms in most cases is within 3 years. The symptoms can also occur as short as 3 months or as long as 8 years. The visual recovery is poor. Some recovery is reported if the treatment is started within 72 hours.

MRI Brain is indicated to differentiate the radiation optic neuropathy from the recurrence of primary brain tumour.

DIABETIC PAPILLOPATHY

It is considered as the variant of NAION. It usually present as unilateral or bilateral disc edema with minimal vision loss and resolves spontaneously without treatment. Optic disc show dilated telangiectatic vessels that can be confused with new vessels of the disc. It is seen in eyes with pre proliferative or proliferative diabetic retinopathy and also in eyes with no evidence of diabetic retinopathy.

COMPRESSIVE OPTIC NEUROPATHY

ANTERIOR COMPRESSIVE OPTIC NEUROPATHY

It usually presents with optic disc edema. Most of the lesions present within the orbit, rarely from intracranial extension. The lesion includes inflammation, infection, tumour etc. The orbital lesion includes optic glioma, meningioma, hemangioma, primary or secondary malignancies, orbital pseudotumour, thyroid orbitopathy, etc.

It usually present with gradual and progressive loss of vision associated with proptosis. The vision loss is near normal or normal with no evidence of external orbital disease.

The grade of RAPD and the color vision loss depends on the amount of compression.The enlargement of blindspot in visual field in the affected eye may present. On fundus examination, unilateral disc edema with various folds in the posterior pole.

Sometimes there may be interruption of blood supply to the optic nerve due to direct pressure which can cause transient monocular visual loss. CT, MRI and ultrasonogram can provide information about the differential diagnosis of compressive optic neuropathy. CT scan is used for imaging bone. MRI defines the intrinsic disease of visual pathway including intracanalicular portion of optic nerve.

In case of orbital pseudotumour, the optic nerve is compressed causing secondary disc edema. The associated features like visual loss, pain, proptosis and congestion can be seen. In case of thyroid orbitopathy, the patient my develop compression of optic nerve due to enlargement of the muscles and fat tissues in the intraconal space. This usually develops in the active phase of thyroid disease with bilateral and symmetric involvement. MRI orbit helps in delineating the optic nerve in case of compression. The treatment includes intravenous or oral steroids. If there is no improvement with steroids, orbital decompression should be done.

Optic nerve sheath meningioma can also lead to optic nerve compression. Sometimes it causes vision loss due to compromise in the blood supply of optic nerve. This tumour occurs in middle aged women and the patient presents with gradual progressive loss of vision with minimal proptosis. On fundus examination, there can be optociliary shunts seen both in swollen and atrophic disc. MRI orbit shows diffuse,

fusiform tumour separated from the optic nerve proper on coronal views. On CT, calcification can be seen and also linear bright lines extending over the length of the optic nerve [tram track sign].

Radiation therapy is the best treatment. Surgical excision does not provide useful vision because of the pial blood vessel involvement. Sometimes intracranial lesions like sphenoidal wing meningioma can extend through the optic nerve canal causing compression.

Retrobulbar optic nerve compression produces painless progressive loss of vision with any type of field defect but it does not cause disc edema.

Ethmoidal mucocoele is a rare cause of compressive optic neuropathy[18]. Painless progressive loss of vision is the commonest symptoms. High degree of suspicion is needed and MRI Orbit is needed to rule out the optic nerve compression[18,19]. Fundus can show an edematous disc or a pale disc due to chronic compression. Urgent endonasal sinus surgery is needed for such patients[20]. De roofing the optic canal is needed to relieve compression [19].

INFILTRATIVE OPTIC NEUROPATHY

The optic nerve is infiltrated by variety of conditions like tumour, infections, inflammation, etc. The lesion includes

LESION	CAUSES
primary tumours	capillary hemangioma, optic glioma,
Secondary tumours	Metastatic carcinoma, lymphoreticular tumours, nasopharyngeal carcinoma
Inflammation	Sarcoidosis, idiopathic perioptic neuritis
Infections	Bacterial, fungal, viral

PRIMARY TUMOURS

OPTIC NERVE GLIOMA

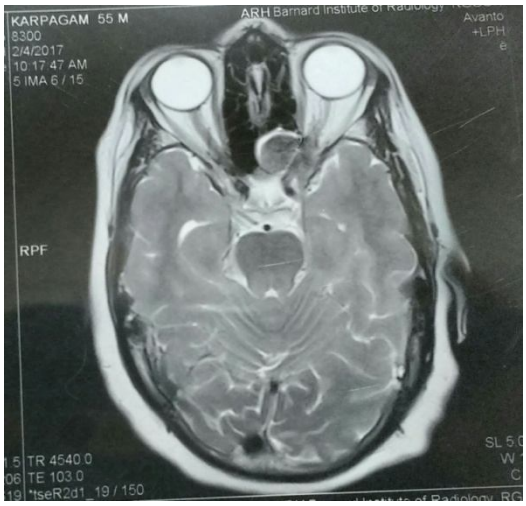
Primary tumours are more common than secondary tumour. Optic nerve glioma is the most common primary tumour represents 1% of all intracranial tumour and 25% of all optic pathway gliomas. Based on the location, size and extent of the tumour the clinical presentation varies.

On MRI brain there is a fusiform enlargement of orbital portion of optic nerve with or without optic canal enlargement. It can be differentiated from the optic nerve meningioma using two important signs. The first is kinking of optic nerve within the orbit and the second is double intensity tubular thickening of the nerve.

28% of optic nerve glioma occurs in association with NF1. So neurofibromatosis type 1 should be ruled out in patients with optic nerve glioma. This benign lesion shows good prognosis with long term useful visual function. In <6 years chemotherapy is the first line of treatment and radiation can be given in older children.

CAVERNOUS HEMANGIOMA

These are isolated well circumscribed and encapsulated lesion within the orbit. This is a unilateral, almost asymptomatic lesion, common in females. It can be seen anywhere along the optic pathway. Mostly produces gradual, progressive loss of vision. If there is hemorrhage within the hemangioma, the vision loss is rapid.



SECONDARY TUMOURS

The common among these secondary tumours are metastatic tumours, lympho reticular malignancies and invasive carcinomas from adjacent site.

In metastatic optic nerve involvement, the visual loss is severe. The disc is swollen with a yellowish white mass protruding from the disc surface and vitreous shows tumour cells mimicking vitritis. The common tumour to metastasis is adenocarcinoma from all parts of the body. In males lung and bowel carcinoma are common, wherein females breast and lung carcinoma are common.

If the metastasis involves the posterior aspect of the optic nerve, there is no disc swelling and the disc appears normal. 50% of the patients can develop vein occlusion.

The common tumour from the nearby site to cause disc infiltration is paranasal sinus tumour, brain tumours etc. But these tumours are associated with cavernous sinus involvement causing diplopia, multiple cranial nerve palsies. The diagnosis can be made easily if the patient was already diagnosed to have a primary tumour. Neuroimaging is essential to differentiate the recurrence of primary from radiation induced optic neuropathy.

Although the prognosis is poor, metastatic tumour shows temporary response to radiation therapy.

Lymphoreticular malignancies may cause infiltrative optic neuropathy in the acute or chronic phase of the disease. The common among them are leukaemia and lymphoma. Only 5% of non Hodgkins lymphoma may show infiltration. Depending upon the location, the

signs and symptoms varies. It can cause slow progressive vision loss or an insidious onset vision loss or an acute loss of vision. Acute lymphoreticular malignancies cause disc infiltration more commonly comparing chronic forms.

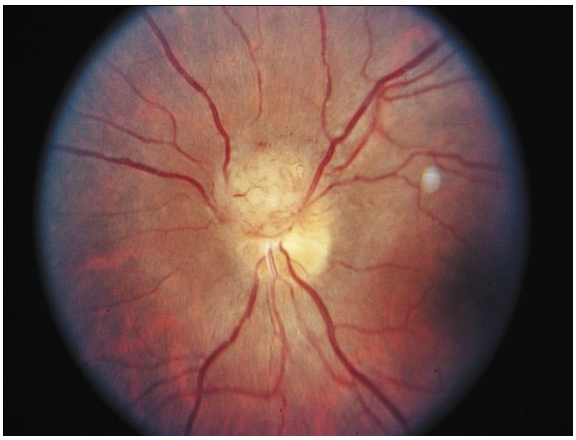
On fundus examination, the disc shows white fluffy infiltrates along with disc edema. Peripapillary and peripheral retinal haemorrhages can also be seen. Diffuse enlargement of the optic nerve with enhancement is seen in neuroimaging. Rapid and good response is seen with radiation therapy and the disc edema is also resolves with the therapy. One should also differentiate the other causes like leukemic retinopathy causing neovascularisation of the disc and the intracranial leukemic infiltrate causing disc edema from infiltrative neuropathy.

Chronic leukaemia presents an indolent course with less severe vision loss.

INFLAMMATORY NEUROPATHY

Sarcoidosis is the most common one causing inflammatory neuropathy. It can affect optic nerve in different ways which includes compression, ischemia, inflammatory and other combined mechanisms. The disc edema is severe and unilateral with dilated vessels over the disc. It is usually associated with other ocular features like anterior segment inflammation, vasculitis [candle wax drippings], intermediate uveitis, vitritis etc.

The diagnosis is made with elevated ACE levels, biopsy of involved tissues [lacrimal gland, conjunctiva] and CT imaging of the chest. The treatment involves steroid along with immnosuppressive.



INFECTIOUS OPTIC NEUROPATHY

Tuberculosis can cause disc edema due to direct involvement or from the inflammation due to adjacent structure involvement. Syphilitic gumma can also cause disc edema due to retrobulbar optic neuritis or

due to gumma within the optic nerve. The viruses like cytomegalo virus and some herpes virus can also cause infiltrative disc edema. The vision loss can be acute or sub acute or chronic.

MANAGEMENT OF UNILATERAL DISC EDEMA

CAUSE OF UNILATERAL DISC EDEMA	INVESTIGATION	TREATMENT	PROGNOSIS
OPTIC NEURITIS	Total count, differential count, ESR, mantoux, chest X-Ray. Neuroimaging is needed if other than demyelinating disease is suspected like inflammatory or compressive neuropathy and in recurrent case of optic neuritis [increased risk of multiple sclerosis]	Intravenous methyl prednisolone for 5 days followed by oral prednisolone for 11 days.	Natural course of the disease not affected by the treatment. But immediate visual recovery with reduced risk of developing multiple sclerosis. Temporal pallor sets in even though the visual recovery is full. Residual defects in colour vision and contrast sensitivity persists
AION	Non Arteritic- RBS BP Lipid profile Arteritic- ESR, CRP Temporal artery biopsy	In nonarteritic type-systemic factors control is the main treatment. In arteritic type-intravenous steroids are the main stay in order to prevent the involvement of the other eye.	Poor prognosis Most cases the vision and field defect does not improve and the disc goes for secondary atrophy in 6-8weeks

Neuroretinitis	Have to rule out infectious and inflammatory causes	Depending upon the cause oral antibiotics or steroids should be started	Good prognosis Optic disc edema resolves and usually the pallor does not sets in.
Compressive optic neuropathy	Imaging –to find out the cause of compression. Retrobulbar mass lesion like cavernous haemangioma, optic nerve glioma and extension from the adjacent [ethmoidal mucocoele]	Immediate relieve of compression by doing FESS[functional endoscopic sinus surgery] or by removing the mass lesion through orbitotomy	Good prognosis with prompt surgical management.
Inflammatory [posterior uveitis] cause of disc edema	Investigations to rule out infectious and inflammatory causes of posterior uveitis TORCH Mantoux test Chest X-Ray VDRL ACE ANA	Oral Antibiotics or steroids depending upon the cause	Good prognosis

PART-II

AIM OF THE STUDY:

To assess the clinical profile of cases of unilateral disc edema.

OBJECTIVES:

1. To assess the causes of unilateral disc edema, age of presentation, sex preponderance, systemic associations, risk factors, treatment and prognosis.
2. To study the clinical profile and categorize the cases of unilateral disc edema with respect to etiology.

MATERIALS & METHODS:

This prospective study was conducted at Squint & Neuro Ophthalmology services, RIO GOH, Egmore, Chennai for a period of 6 months [March 2017 to August 2017].

INCLUSION CRITERIA:

1. Patients presenting with unilateral disc edema.
2. Both males and females of age 20 - 65 years.

EXCLUSION CRITERIA:

1. Patient with bilateral presentation and papilledema.
2. Age < 20 years.

EVALUATION OF PATIENTS:

Patients presenting to Squint & Neuro Ophthalmology services were registered, evaluated and followed up during the study period.

- Detailed history of present illness.
- Visual acuity was measured using Snellen's acuity chart and converted to logmar for the purpose of statistical analysis.
- Slit lamp bio microscopy of anterior segment, fundus with +90D lens.
- Intraocular pressure.
- Direct and Indirect Ophthalmoscopy.
- Fields using automated perimetry.
- Colour vision using ishihara chart.
- Blood investigations (Total count, Differential count, Erythrocyte sedimentation rate, VDRL, Mantoux, Blood sugar, Fasting lipid profile).
- Blood pressure.
- Radiological imaging [chest X-Ray, MRI Brain, Orbit, Spine (if needed)].

FOLLOW UP PROCEDURES/VISITS:

1^{st}, 2^{rd}, 4^{th} week, 3^{rd} and 6^{th} month

ASSESSMENTS OF PARAMETERS:

- Improvement in visual acuity.
- Fundus examination.
- Colour vision.
- Fields.

RESULTS

Statistical analysis plan-Data analysis

Data collected were entered in Excel Spread sheet and analyzed using STATA statistical software package release 11. We used the two-sided independent-samples t test to compare means across dichotomous variables (i.e. men v. women); the one-way ANOVA test for comparison of means across multilevel variables. Simple calculations like Percentages, Proportions and Mean values were derived. A type I error of 0.05 was considered in all analyses.

MANAGEMENT

INVESTIGATIONS DONE:

All the patients presented with unilateral disc edema are subjected to following investigation

- A detailed history of the patient,
- Slit lamp examination,
- Fundus examination using +90D lens and with both direct and indirect ophthalmoscopy,
- Best corrected visual acuity,
- Colour vision,
- Fields and
- Blood investigations including Total count, Direct count, Erythrocyte sedimentation rate, Mantoux, VDRL, Blood sugar, Fasting lipid profile and

- Neuroimaging(MRI brain and MRI Spine) was done only if the patient was high risk for multiple sclerosis or the patient was a suspect of Devics disease.
- MRI Orbit to rule out compressive optic neuropathy.
- Blood pressure was checked for all patients.
- Patients were managed depending upon their causes.
- Best corrected visual acuity, colour vision, fields, Slit lamp examination, fundus examination were done during their follow-up period.

MEDICAL MANAGEMENT

The treatment varies depending upon the cause of unilateral disc edema

OPTIC NEURITIS

Patients with unilateral disc edema diagnosed as optic neuritis are treated with intravenous methyl prednisolone for 3 days followed by oral prednisolone for 11 days as per ONTT [optic neuritis treatment trial] along with neurovitamins

The patients were subjected to other investigations like CSF Analysis, neuroimaging[MRI Brain and Spine], NMO antibodies only if,

- It is a recurrent case of optic neuritis [To rule out the risk of multiple sclerosis]
- It is associated with other features like periphebitis or intermediate uveitis [Has high risk for multiple sclerosis]

- If the patient, had a past history of paraplegia. [To rule out neuromyelitis optica]

AION [all cases of AION in our study are NAION]

In patients with age >40 year

- Systemic causative factors like diabetic, hypertension, hyperlipaedemia are ruled out by doing FBS, BP, fasting lipid profile.
- Other investigations like carotid Doppler and cardiac evaluation were also done to rule out carotid or cardiac emboli.
- Patients with systemic factors like uncontrolled diabetes and hypertension are advised to control systemic factors.
- Oral prednisolone was started for patients with no systemic factors

In patients less than 40 years

In addition to FBS, BP and Lipid profile patients were also advised to do coagulation profile, homocysteine levels [to rule out hypercoagulable states], ESR, RA factor, ANA were done [to rule out the vasculitic cause of AION].

Neuroretinitis

It is subdivided into infectious and inflammatory causes and the patient treatment is based on the cause. Patients were subjected to do

complete blood count, differential count, ESR, VDRL, Mantoux, chest X-Ray.

Inflammatory causes were treated with steroids and infectious causes of neuroretinitis were treated with antibiotics initially for few days and followed by antibiotics along with steroids.

Inflammatory disc edema

In patients with inflammatory disc edema [posterior uveitis causing disc edema],

Detailed slit lamp examination [to rule out anterior chamber reaction and vitritis]

Detailed direct and indirect ophthalmoscopic examination of fundus [to rule out choroiditis patch, peripheral toxoplasmosis lesion etc]

Patients were also subjected to do complete blood count, differential count, ESR, VDRL, Mantoux, chest X-Ray to find out the cause for disc edema

Depending upon the cause for disc edema, the patients were treated. Inflammatory causes were treated with steroids and infectious causes of disc edema were treated with antibiotics initially for few days and followed by antibiotics along with steroids.

SURGICAL MANAGEMENT

Urgent surgical intervention was done for patients presented with compressive disc edema.

RESULTS AND ANALYSIS

CAUSES OF UNILATERAL DISC EDEMA

DIAGNOSIS	Frequency	Percentage
NAION	16	53.33
COMPRESSIVE NEUROPATHY	3	10
INFLAMMATORY NEUROPATHY	3	10
NEURORETINITIS	3	10
OPTIC NEURITIS	5	16.67
Total	30	100

Table: 1 showing causes of unilateral disc edema

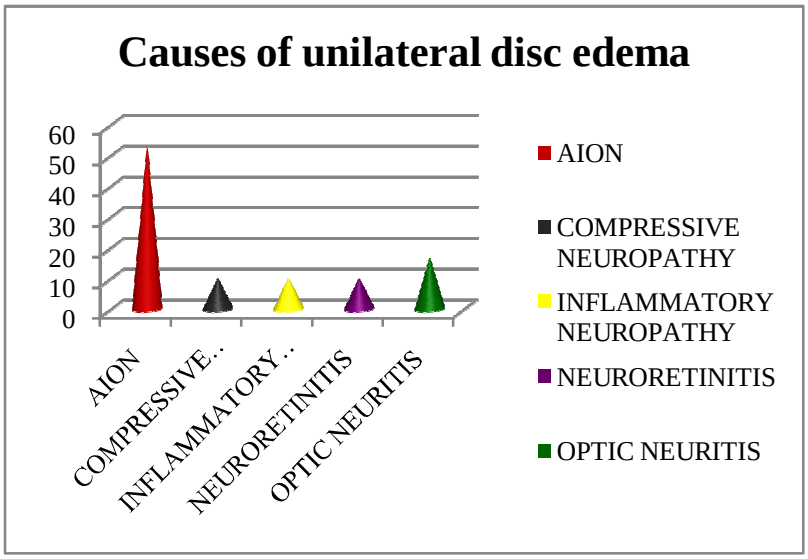


Chart 1: Bar diagram showing various causes of unilateral disc edema

It is evident from the study, the commonest cause for unilateral disc edema is nonarteritic AION and the next common cause is optic

neuritis. Compressive disc edema, inflammatory disc edema and neuroretinitis are the other conditions causing unilateral disc edema.

AGE DISTRIBUTION IN CASES OF UNILATERAL DISC EDEMA

Age	Observations	Mean	SD	Min	Max
AION	16	49.00	10.04	34	74
COMPRESSIVE NEUROPATHY	3	52.33	5.69	46	57
INFLAMMATORY [POSTERIOR UVEITIS] DISC EDEMA	3	31.67	8.02	24	40
NEURORETINITIS	3	52.67	15.14	42	70
OPTIC NEURITIS	5	28.40	5.68	22	35

Table: 2 Age distribution in cases of unilateral disc edema in the study group

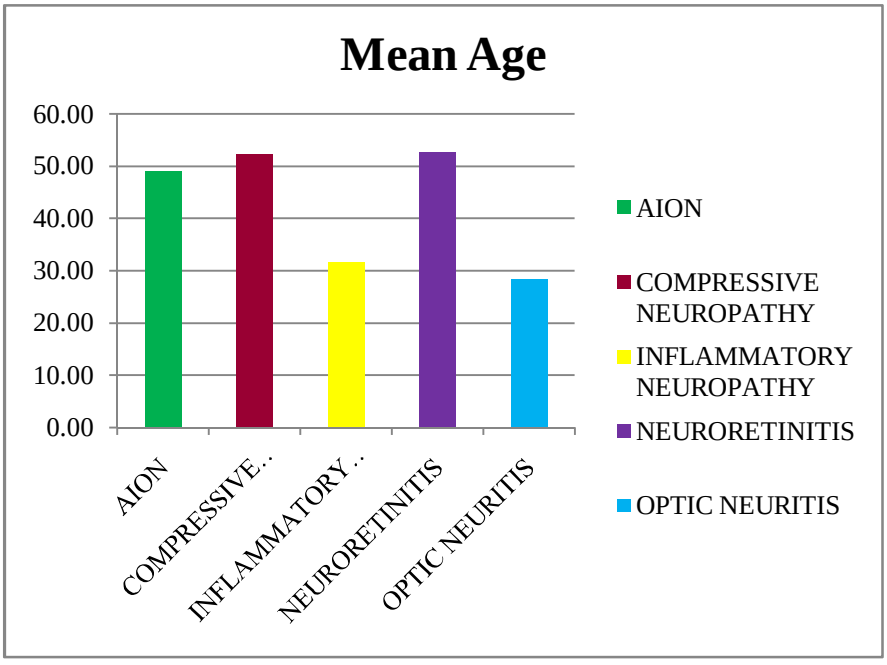


Chart 2: Bar diagram age distribution in cases of unilateral disc edema in the study group

The mean age of presentation of NAION is 49 and the mean age of presentation of optic neuritis is 28. This indicates the association of systemic disease with NAION as it affects the age group between 40 to 50 years and the association of demyelination with optic neuritis as it affects the age group between 20 to 30 years.

SEX DISTRIBUTION IN GENERAL

SEX [in general]	Observation	Percent
Women	17	56.67
Men	13	43.33
Total	30	100

Table 3: Sex distribution in general in the study group

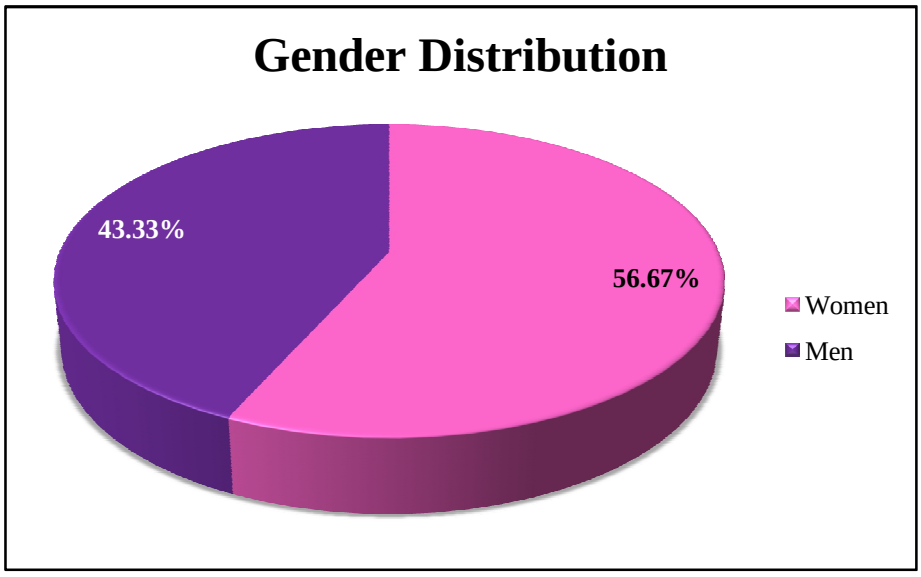


Chart 3: Sex distribution in general in the study group

In general females are affected more commonly compared to males in the study group.

LATERALITY

AFFECTED EYE	Freq.	Percent
Left	15	50
Right	15	50
Total	30	100

Table 4: Laterality in general in the study group

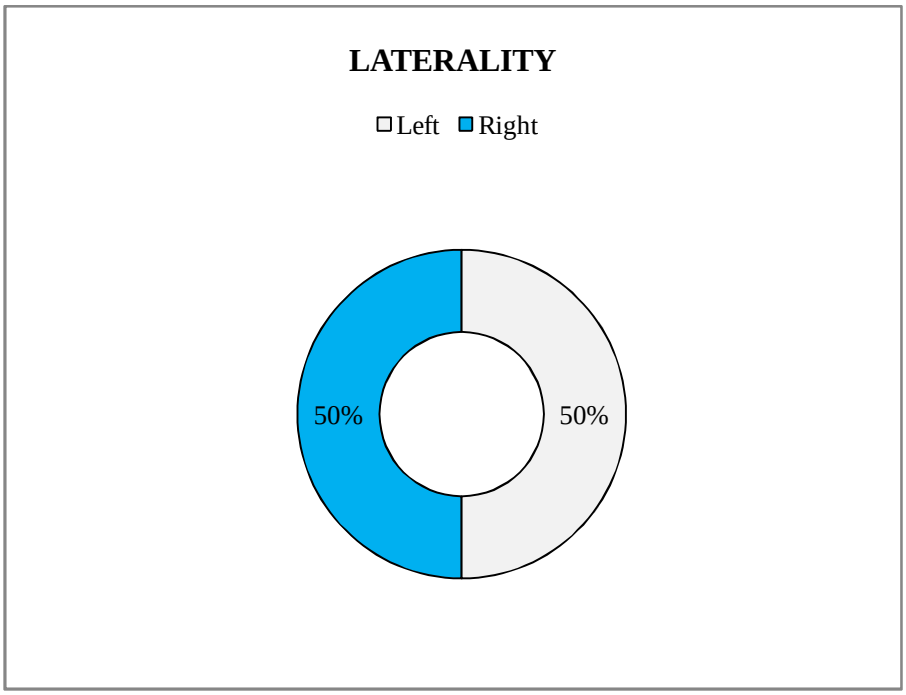


Chart 4: Laterality in general in the study group

All the cases in our study had unilateral affection of the disease. There is no specificity of the eye involved. Both eyes are equally affected in the study group.

PUPILLARY REACTION IN VARIOUS CAUSES OF DISC EDEMA

PUPIL	AION		OPTIC NEURITIS		COMPRE-SSIVE NEURO-PATHY		INFLAM - MATORY DISC EDEMA		NEURORE-TINITIS		TOTAL	
	N	%	N	%	N	%	N	%	N	%	N	%
NORMAL	0.0	0.0	0.0	0.0	0.0	0.0	2.0	66.7	3.0	100.0	5.0	16.7
GI RAPD	9.0	56.3	1.0	20.0	2.0	66.7	0.0	0.0	0.0	0.0	12.0	40.0
GII RAPD	6.0	37.5	1.0	20.0	0.0	0.0	0.0	0.0	0.0	0.0	7.0	23.3
GIII RAPD	0.0	0.0	0.0	0.0	1.0	33.3	0.0	0.0	0.0	0.0	1.0	3.3
GIV RAPD	0.0	0.0	3.0	60.0	0.0	0.0	0.0	0.0	0.0	0.0	3.0	10.0
SRL	1.0	6.3	0.0	0.0	0.0	0.0	1.0	33.3	0.0	0.0	2.0	6.7
Total	16.0	100.0	5.0	100.0	3.0	100.0	3.0	100.0	3.0	100.0	30.0	100.0

Table 5: Pupillary reaction in various causes of disc edema

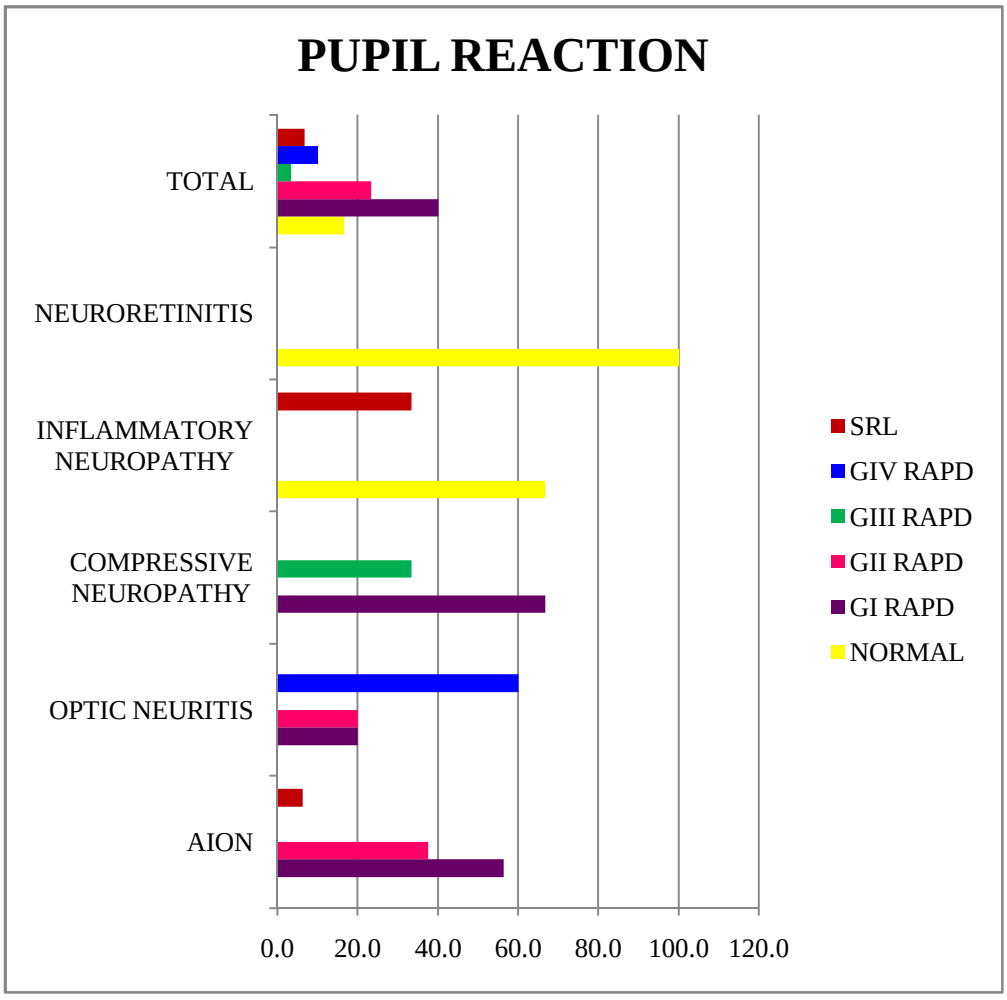


Chart 5: Pupillary reaction in various causes of disc edema

In our study RAPD is present in all cases of optic neuritis and almost in all cases of ischemic optic neuropathy except one. The severity of RAPD is more in optic neuritis compared to NAION. 93.5% of NAION patients have grade I to II RAPD whereas 100% of patients with optic neuritis have grade III to IV RAPD.

66.7% of inflammatory disc edema has normal pupillary response and 100% of neuroretinitis cases have normal pupillary reaction.

FUNDUS FINDINGS IN UNINVOLVED EYE

OTHER EYE FUNDUS	Frequency	Percent
ARTERIOLAR ATTENUATION	6	20
DISC AT RISK	2	6.67
GIII HT RETINOPATHY	1	3.33
HIGH MYOPIC	1	3.33
NORMAL	15	50
NO VIEW	1	3.33
DM RETINOPATHY	2	6.67
PALLOR	1	3.33
ROTH SPOTS	1	3.33
Total	30	100

Table 6: Fundus findings in uninvolved eye

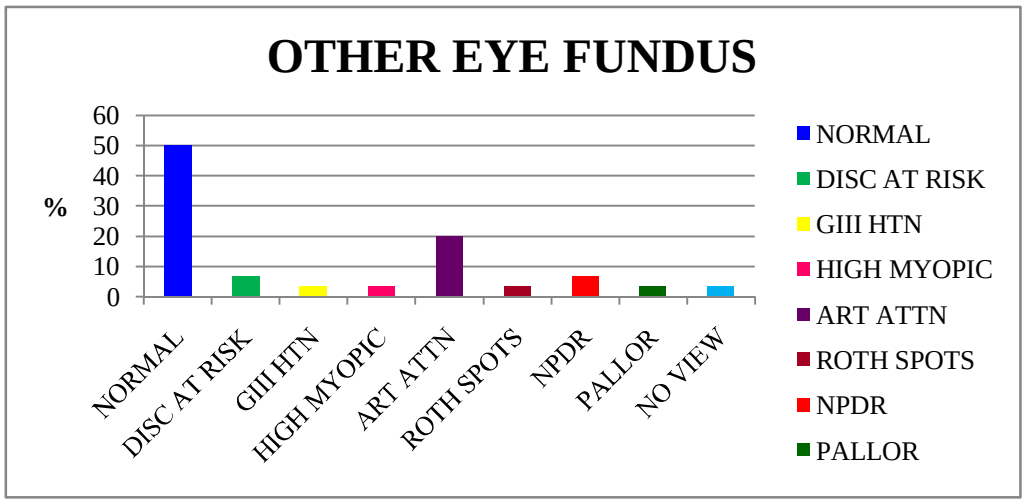


Chart 6: Fundus findings in uninvolved eye

In this study, Fundus examination of uninvolved eye showed normal fundus in 50% of cases. Other 50% of cases showed some findings which helped in diagnosing the condition. 'At risk' crowded disc was seen in 6.67% of persons, hypertensive and diabetic changes were noted in 10% of cases.

DISC PALLOR ON FOLLOW UP

DISC PALLOR ON FOLLOW UP	PRESENT		ABSENT	
	FREQ	PERCENT	FREQ	PERCENT
AION	12	66.67	4	33.33
COMPRESSIVE OPTIC NEUROPATHY	1	5.56	2	16.67
NEURORETINITIS	0	0.00	3	25.00
OPTIC NEURITIS	5	27.78	0	0.00
INFLAMMATORY DISC EDEMA	0	0.00	3	25.00
TOTAL	18	100	12	100

Table 7: Disc pallor on follow up in various causes of unilateral disc edema

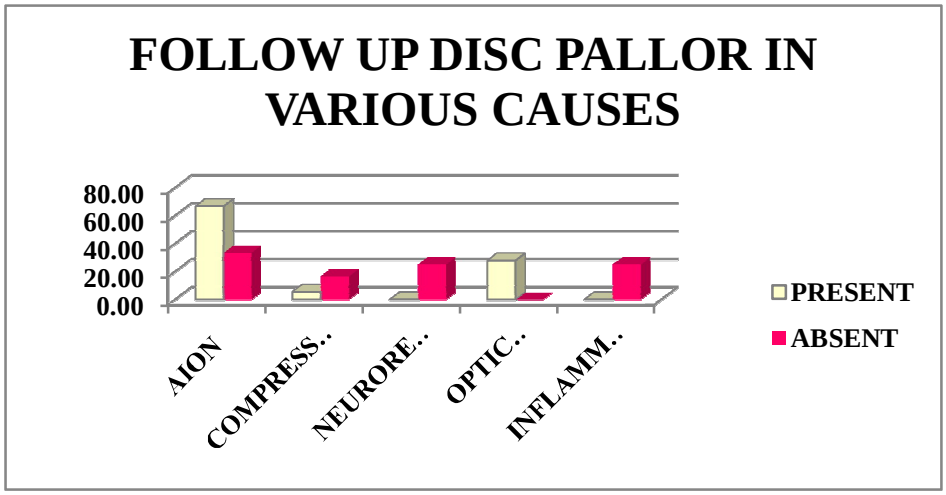


Chart 7: Disc pallor on follow up in various causes of unilateral disc edema

In this study 60 % of the persons showed disc pallor on follow up and 40 % of person showed normal fundus. 66.67 % of patients of NAION had disc pallor on follow up. All the patients of optic neuritis had disc pallor on follow up even though the vision was good. Neuroretinitis disc edema and inflammatory disc edema usually resolved without producing any disc pallor.

IMAGING IN UNILATERAL DISC EDEMA

IMAGING IN UNILATERAL DISC EDEMA	**Frequency**	**Percent**
MRI Normal	2	6.67
MRI SPINE LETM[TRANSVERSE MYELITIS]	1	3.33
MRI ETHMOIDAL MUCOCELE	1	3.33
MRI OPTIC NEURITIS	3	10
MRI SCLERITIS	1	3.33
MRI BRAIN SMALL VESSEL ISCHEMIA	2	6.67
MRI ORBIT EXTRACONAL MASS	1	3.33
MRI ORBIT INTRACONAL HAEMANGIOMA	1	3.33
Not Done	18	60
Total	30	100

Table 8: Imaging in unilateral disc edema

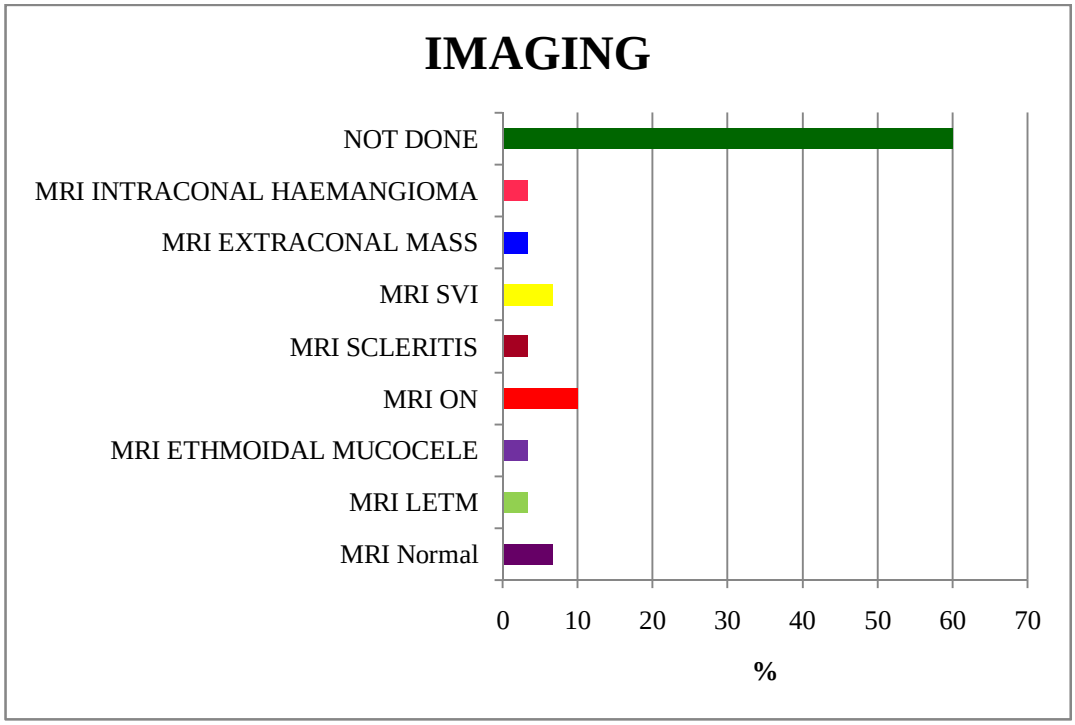


Chart 8: Imaging in unilateral disc edema

MRI imaging was needed only in 40 % of the patients. It was 100% useful in compressive neuropathy and also aided in diagnosing optic neuritis in some patients. It also helped in diagnosing longitudinally extensive transverse myelitis which was an important sign in diagnosing neuromyelitis optica. MRI BRAIN in NAION showed small vessel ischemic changes in 6.67% of persons which indicates the ischemic change were also noted in brain.

OTHER INVESTIGATIONS DONE IN UNILATERAL DISC EDEMA

OTHER INVESTIGATIONS	Frequency	Percent
AQ4 POSITIVE	1	3.33
PT, INR – NORMAL	1	3.33
TORCH +	2	6.67
NOT DONE	26	86.67
Total	30	100

Table 9: Other investigations done in unilateral disc edema

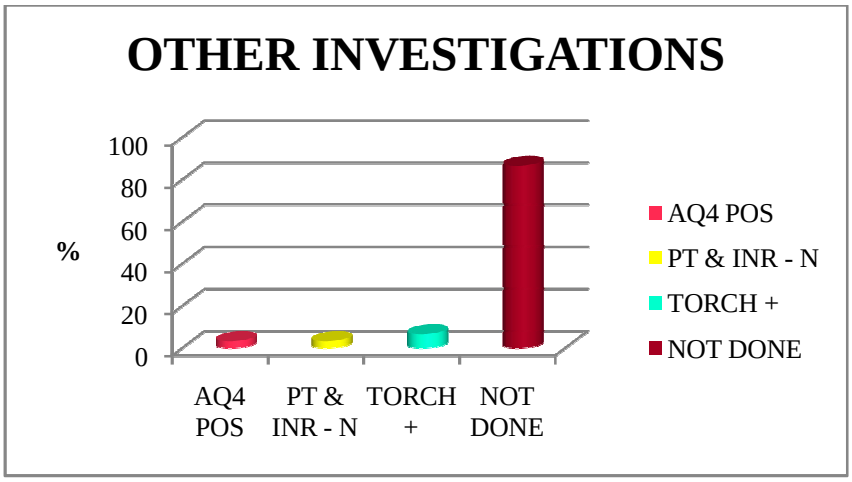


Chart 9: Other investigations done in unilateral disc edema

In our study other special investigations were done in only 12.33% of the patients. These investigations are ordered depending upon the clinical signs and symptom of patients with unilateral disc edema. Serum Aquaporin- 4 antibodies was done in 3.33% of patients with disc edema to rule out neuromyelitis optica. TORCH screening was done in 6.67% of the patients with disc edema to rule out infectious cause of disc edema. PT INR was done in 3.33% of patient with disc edema to rule out hypercoagulable cause of AION.

As NAION was the most common cause of unilateral disc edema in this study, NAION parameters are elaborated in detail in the following results and analysis part.

AGE DISRIBUTION IN NAION

AGE GROUPS IN NAION	Frequency	Percent
30 - 39 yrs	2	12.5
40 - 49 yrs	8	50
50 - 59 yrs	3	18.75
60 - 69 yrs	2	12.5
≥70 yrs	1	6.25
Total	16	100

Table10: Age distribution in NAION

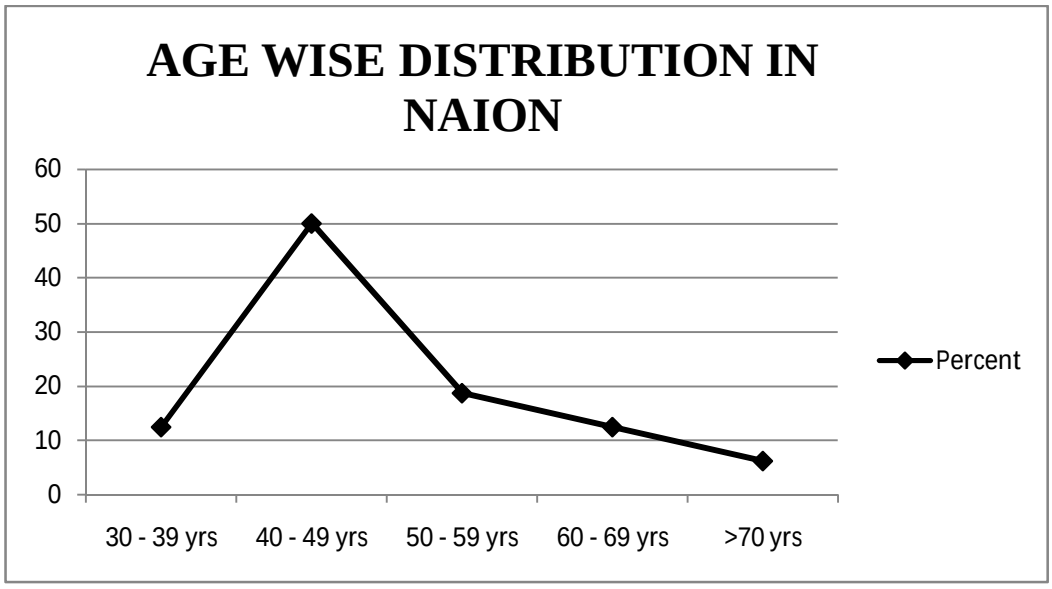


Chart 10: Age distribution in NAION

In our study, the most common age group to get affected in NAION is 40 to 50 years group. The next common is 50 to 60 years

group[21]. This may be due to increased association of causative factors like DM and HT in this age group.

SEX DISTRIBUTION IN NAION

Gender in NAION	Frequency	Percent
Women	4	25
Men	12	75
Total	16	100

Table 11: Sex distribution in NAION

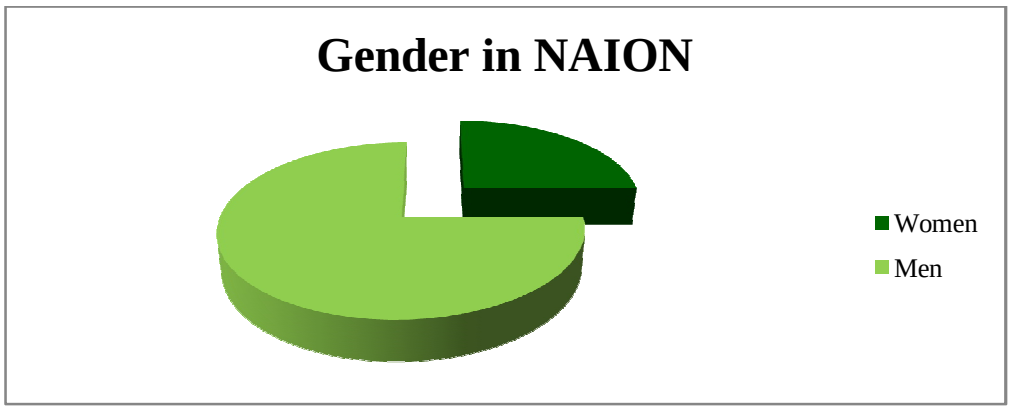


Chart 11: Sex distribution in NAION

Males are more commonly affected by NAION in our study. This may be due to the increased association of risk factors in males in this age group compared to females of the same age.

FUNDUS CHANGES IN NAION

FUNDUS CHANGES IN NAION		Frequency	Percentage
MEDIA	CLEAR	15	93.75
	PCO	1	6.25
MARGIN	BLURRED	16	100
	NORMAL	0	0
HYPEREMIA	ABSENT	1	6.25
	PRESENT	15	93.75
DISC HEMORRHAGE	ABSENT	1	6.25
	PRESENT	15	93.75
SECTORAL PALLOR	ABSENT	0	0
	PRESENT	16	100
OTHER FINDINGS	ART. ATTENUATION	37.5	37.5
	GIII HT RETINOPATHY	1	6.25
	DIABETIC RETINOPATHY	3	18.75
	NO	3	18.75
	VENOUS DILATATION	2	12.5
OTHER EYE FUNDUS	DISC AT RISK	2	12.5

Table 12: Fundus changes in NAION

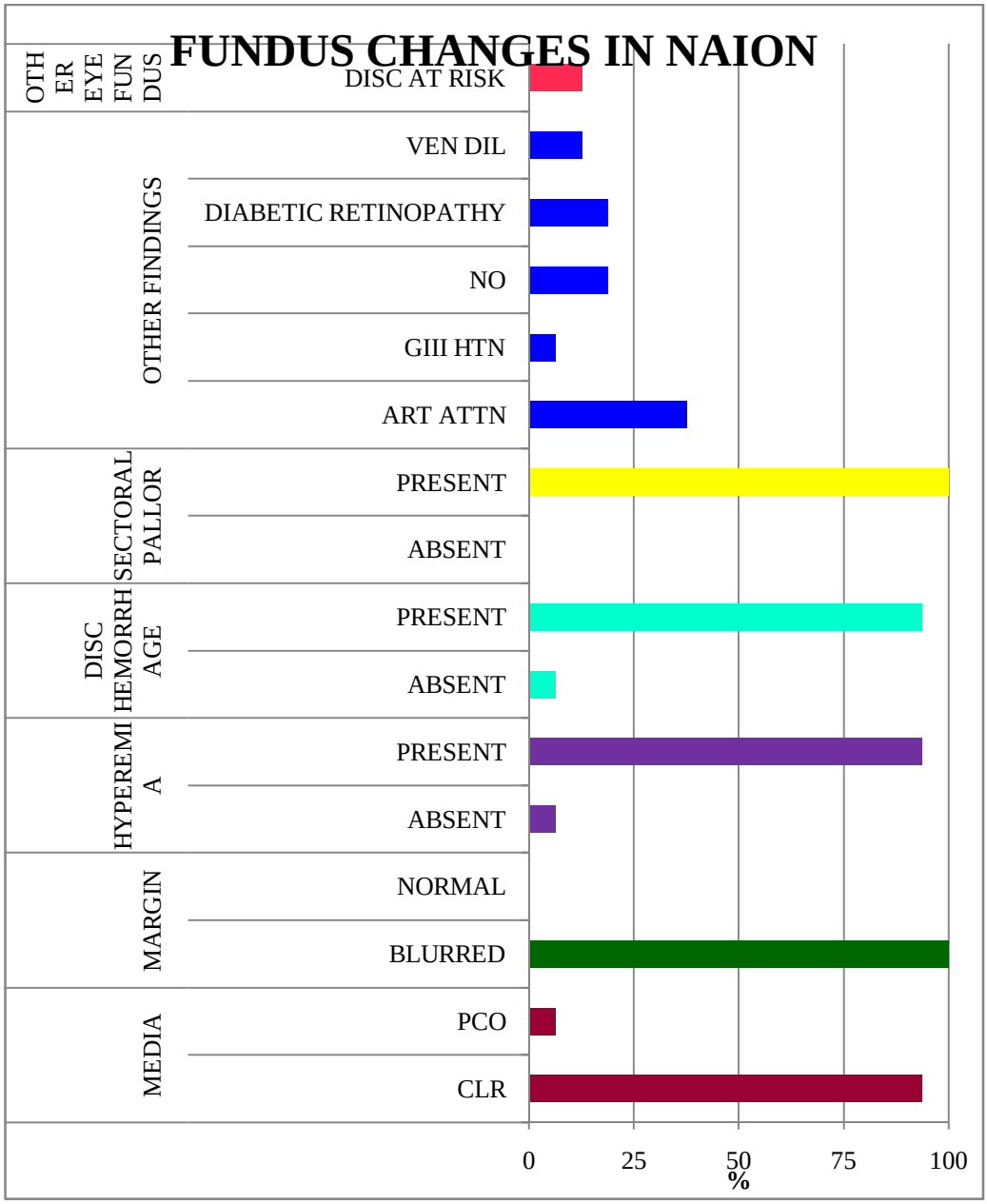


Chart 12: Fundus changes in NAION

The commonest fundus changes in NAION are sectoral disc pallor, hyperemia and haemorrhages in the peripapillary region in our study. Other associated findings like diabetic and hypertensive changes are noted in 10 % of persons with NAION. Other eye fundus finding like crowded disc is seen in 12.5% of persons with NAION.

FUNDUS CHANGES IN OPTIC NEURITIS

FUNDUS CHANGES IN OPTIC NEURITIS		**Frequency**	**Percentage**
MEDIA	CLR	5	100
	OTHERS	0	0
MARGIN	BLURRED	5	100
	NORMAL	0	0
HYPEREMIA	ABSENT	0	0
	PRESENT	5	100
DISC HEMORRHAGE	ABSENT	4	80
	PRESENT	1	20
SECTORAL PALLOR	ABSENT	5	100
	PRESENT	0	0
OTHER FINDINGS	NO	4	80
	VITRITIS	1	20
OTHER EYE FUNDUS	NORMAL	4	80
	PALLOR	1	20

Table 13: Fundus changes in optic neuritis

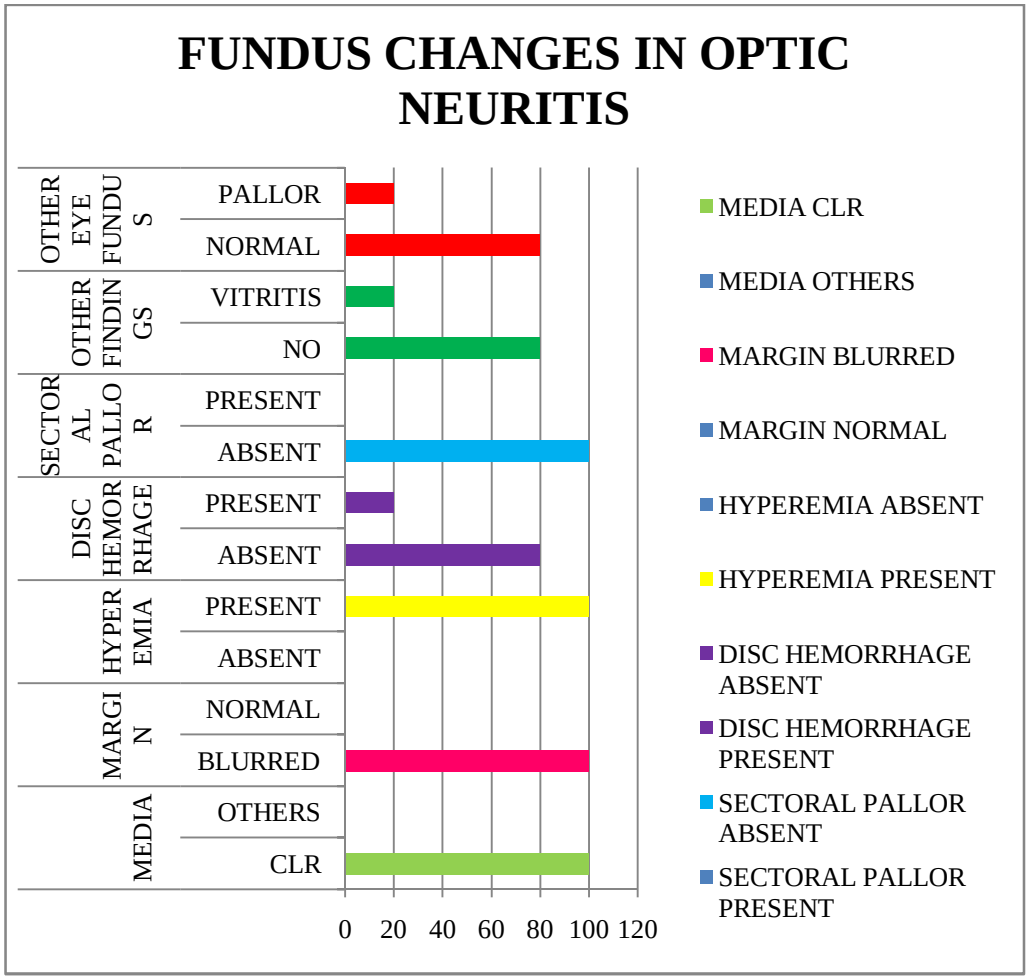


Chart 13: Fundus changes in optic neuritis

In our study, the most common fundus finding in optic neuritis was hyperemia and was seen in 100 % patients and sectoral pallor was absent in all patients with optic neuritis. Other findings like disc haemorrhages and vitritis were seen in 20% of optic neuritis patient.

FIELD CHANGES IN NAION

FIELD DEFECTS IN NAION	Frequency	Percentage
NORMAL	1	6.25
INF. QUADRANTANOPIA	1	6.25
INF. ALTITUDINAL DEF.	5	31.25
PARACENT SCOTOMA	1	6.25
NOT POSSIBLE	8	50
Total	16	100

Table 14: Field changes in NAION

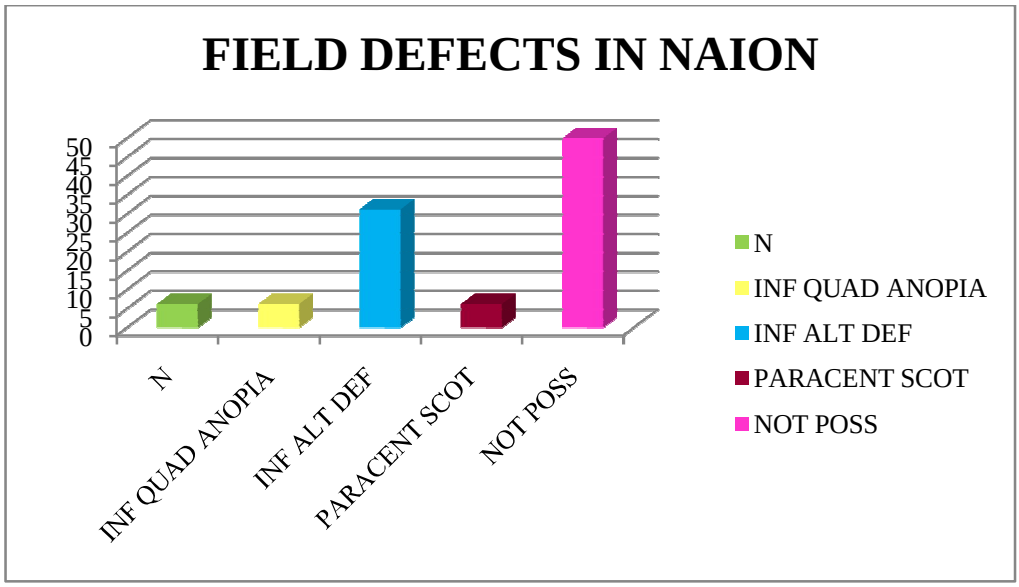


Chart 14: Field changes in NAION

In our study 50% of persons were not able to do field charting due to poor vision. In the remaining 50% of persons, the most common field defect was inferior altitudinal defect. Paracentral scotoma was rare to present and was seen in 6.25% of persons[22].

ASSOCIATION OF SYSTEMIC IINESS WITH NAION

SYSTEMIC ILLNESS IN NAION	FREQUENCY	PERCENTAGE
NIL	5	31.3
DM	4	25
HTN	2	12.5
Dm + HTN	5	31.3
Total	16	100

Table 15: Association of NAION with systemic illness

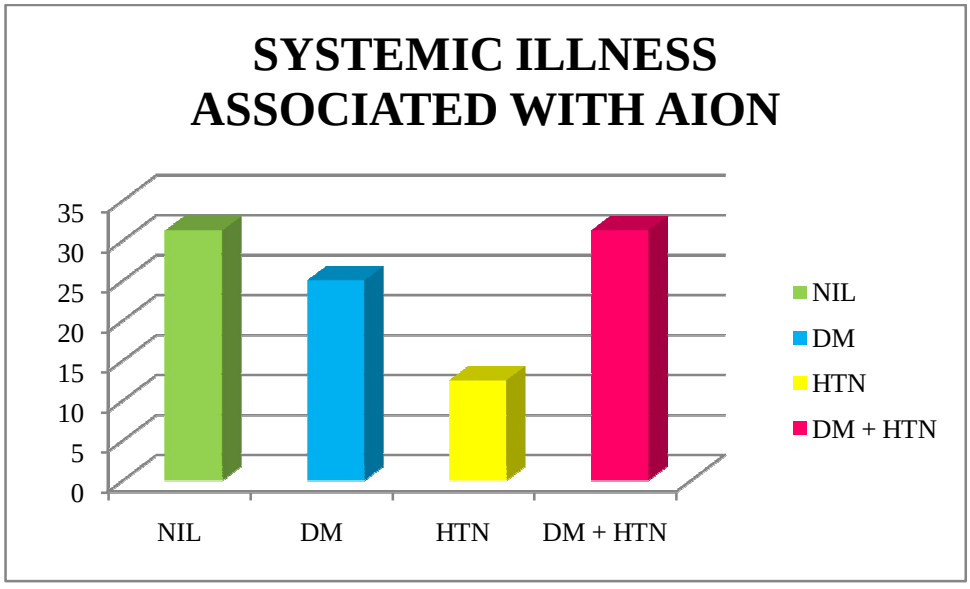


Chart 15: Association of NAION with systemic illness

In this study 68.8% of the patients with NAION had associated systemic illness. Out of that 68.8%, 25% had diabetes mellitus, 12.5% had hypertension and 31.3% had both DM and HT.

This indicates NAION had high risk of having associated systemic illness and all NAION should undergo blood investigations to rule out systemic illness like DM, HT and hyperlipidaemia[23,24,25].

CAROTID DOPPLER STUDY IN NAION

CAROTID DOPPLER IN NAION	Frequency	Percentage
NORMAL	15	93.75
NON SIG STENOSIS	1	6.25
Total	16	100

Table 16: Carotid doppler study in NAION

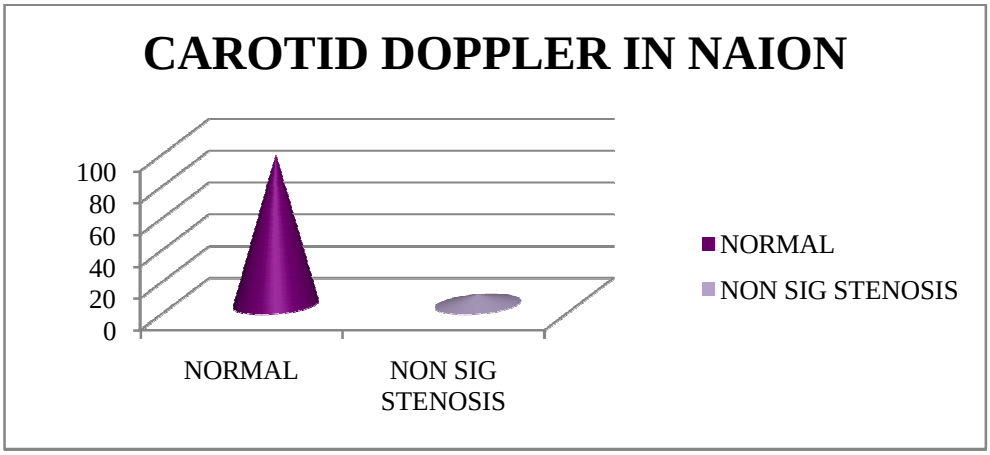


Chart 16: Carotid doppler study in NAION

In our study, 93.75% of patients show normal carotid Doppler. Only 6.25% of persons have insignificant stenosis on the contralateral side in the carotid Doppler. This infers that the carotid doppler is not must for all cases and it can be tailored to patients with signs and symptoms of occlusion like pain and bruit. The association of carotid artery atherosclerosis is very uncommon with NAION[25].

FELLOW EYE INVOLVEMENT IN NAION

OTHER EYE INVOLVEMENT IN NAION	Frequency	Percent
YES	2	12.5
NO	14	87.5
Total	16	100

Table 17: Fellow eye involvement in NAION

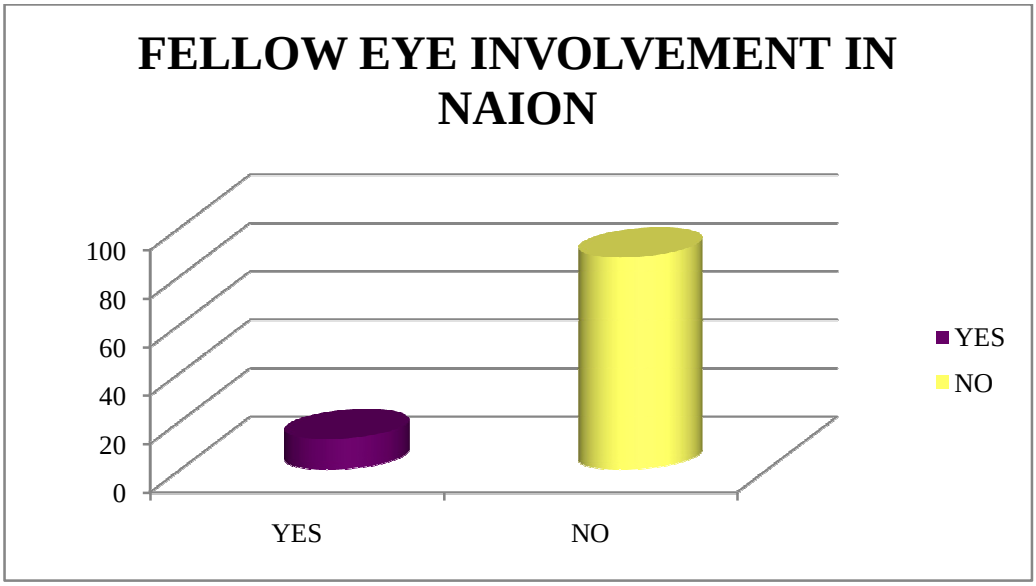


Chart 17: Fellow eye involvement in NAION

In this study, the fellow eye is involved in 12.5% of persons with NAION[27]. This indicates only small percentage of patient with NAION have risk of involvement of the fellow eye.

CHARACTERISTICS OF NEURORETINITIS IN THIS STUDY

NEURO RETINITIS	CASE 1	CASE 2	CASE 3
AGE	70	42	46
SEX	F	F	F
AFFECTED EYE VISION	CFCF	6/60	1/60
AFFECTED EYE PUPIL	RTL	RTL	RTL
TREATMENT	ANTIBIOTIC + STEROID	ANTIBIOTIC + STEROID	ANTIBIOTIC+ STEROID
VN 1WEEK	2/60	6/36	6/60
VN AT 6TH MONTH FOLLOWUP	6/18	6/6	6/12
FOLLOWUP DISC PALLOR	NO	NO	NO

P Value: 0 week to 1st week >0.05

0 week to 6th month <0.05

Table 18: Characteristics of neuroretinitis in this study

Only three cases of neuroretinitis were presented to our clinic. In that all the three were females. All the there showed good visual recovery without any evidence of disc pallor[7].

CHARACTERISTICS OF INFLAMMATORY DISC EDEMA IN THIS STUDY

INFLAMMATORY DISC EDEMA	CASE 1	CASE 2	CASE 3
AGE	31	24	40
SEX	F	F	F
REVN	6/6	CFCF	1/60
REPUPIL	RTL	SRL	RTL
OTHERFINDINGS	TOXOPLASMOSIS	CHOROIDITIS MACULA	PANUVEITIS
TREATMENT	ANTIBIOTIC + STEROID[LATER]	ANTIBIOTIC + STEROID[LATER]	ANTIBIOTIC + STEROID[LATER]
VN 1WEEK	6/6	6/60	6/60
VN AT 6TH MONTH FOLLOWUP	6/6	6/6	6/60
FOLLOWUPDISCPALLOR	NO	NO	NO

P Value: 0 week to 1st week >0.05

0 week to 6th month >0.05

Table 19: Characteristics of inflammatory disc edema in this study

Out of the three cases of inflammatory [posterior uveitic] disc edema, the first was due to toxoplasmosis, 2nd was due to choroiditis of macula and the 3rd was due to panuveitis. All the three cases showed good visual recovery without any disc pallor[28].

CHARACTERISTICS OF COMPRESSIVE OPTIC NEUROPATHY IN THIS STUDY

COMPRESSIVE OPTIC NEUROPATHY	Case 1	Case 2	Case 3
AGE	54	46	57
SEX	F	F	F
LEVN	1/60	6/18	6/36
LEPUPIL	GRADE III RAPD	GRADE I RAPD WITH PROPTOSIS	GRADE I RAPD WITH PROPTOSIS
CAUSE	ETHMOIDAL MUCOCELE	INTRACONAL HAEMANGIOMA	EXTRACONAL MASS
TREATMENT	DECOMPRESSION	ORBITOTOMY MASS REMOVAL	ORBITOTOMY MASS REMOVAL
VN 1WEEK	CFCF	6/12	6/36
VN FOLLOWUP	NO PL	6/12	6/12

P Value: 0 week to 1st week >0.05

0 week to 6th month >0.05

Table 20: Characteristics of compressive optic neuropathy in this study

Out of three cases of compressive neuropathy, first case was due to ethmoidal mucocele, second case was due to cavernous haemangioma and the third case was due to extra conal mass lesion. Among the three cases, ethmoidal mucocele showed poor prognosis as the patient did not have features like pain or proptosis and thereby delay in the management. Eventhough she had undergone endonasal sinus decompression, but the visual recovery was poor[20,19]. But the other two cases showed proptosis which aided in early management and thereby good visual recovery.

VISION IMPROVEMENT IN LOG MAR IN NAION

Mean Difference in Vision in AION	Observation	Mean	Std. Dev.	Min	Max
0 Week	16	1.39	0.96	0	3
1 Week	16	1.30	0.90	0	3
6 Months	16	1.19	1.00	0	3

P Value: 0 week to 1st week >0.05

0 week to 6th month >0.05

Table 21: Vision Improvement in Log MAR in AION

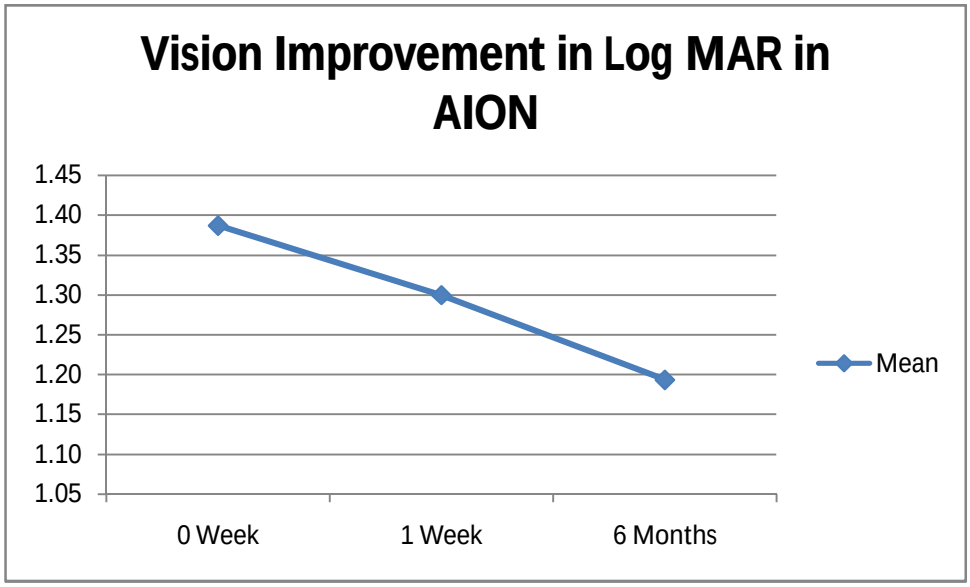


Chart 18: Vision Improvement in Log MAR in AION

The visual improvement in NAION in log MAR was just 0.02 lines with the p- value >0.05 which was a less significant improvement. This indicates the poor prognosis of ischemic optic neuropathy.

VISION IMPROVEMENT in log MAR in OPTIC NEURITIS

Variable	Observation	Mean	Std. Dev.	Min	Max
Vn 0 WEEK	2	2	1.41	1	3
Vn 1 WEEK	5	1.12	0.52	0.6	2
Vn 6 MONTH	5	0.36	0.50	0	1

Table 22: Vision improvement in log mar in optic neuritis

P Value: 0 week to 1st week <0.05

0 week to 6th month <0.01

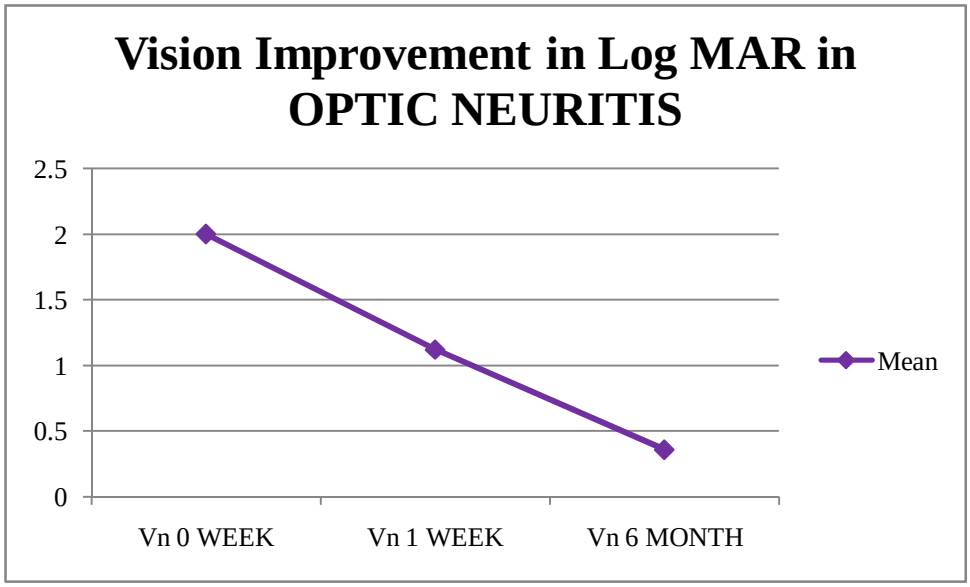


Chart 19: Vision improvement in log mar in optic neuritis

The visual recovery in optic neuritis was excellent at 6th month follow up with the p-value <0.01.

DISCUSSION

Jong Jin Jung, Seung-Hee Baek and et al conducted the study called Analysis of the Causes of Optic Disc Swelling and its result showed that the most common cause with optic disc edema was NA-AION and the second most common cause was ON. There was no case of arteritic AION in this study. The NA-AION was diagnosed at an older age in this study and the common type of field defect in NA-AION was an inferior altitudinal defect. Optic neuritis was associated with a better prognosis than NA-AION. The compressive optic neuropathy causing disc edema is only 6.1%.

In our study also NAION is the most common cause of unilateral disc edema and the second is optic neuritis. Only non arteritic type of AION is presented to our clinic. Even though 1 patient is suspected to have AAION based on her elevated ESR levels, she is not having general symptoms and signs suggestive of arteritis. Like the above study, inferior altitudinal defect is the most common field defect associated with NAION and optic neuritis has better prognosis compared to NAION. In this study compressive disc edema is seen in 10% of the patients compared to the above study which is 6.1%.

Hayreh SS, Zimmerman B conducted a study and assessed the **v**isual field abnormalities in nonarteritic anterior ischemic optic neuropathy: their pattern and prevalence at initial examination. The

study showed an absolute inferior nasal visual field defect is more common (22.4%) than an absolute inferior altitudinal visual field defect (8.0%) in NA-AION and they also found that a combination of relative inferior altitudinal defect with an absolute inferior nasal defect is usually the common pattern seen in NA-AION.

In our study, the inferior altitudinal defect is seen in 31.25% of NAION, the inferior quadrantanopia and paracentral scotoma are seen in 6.25% of NAION. This is comparable to the above study conducted by Hayreh SS, Zimmerman B

c l fry, j e carter, et al studied the relationship between anterior ischemic optic neuropathy and carotid artery atherosclerotic disease to determine if they had an increased occurrence of carotid artery stenosis. They concluded that AION is not associated with carotid artery atherosclerosis in most of the patients. In this study also there was no association between NAION and carotid artery stenosis.

Preechawat P[1], Bruce BB, et al studied the characteristics of AION in patients younger than 50 years. They concluded that AION in younger patients is not uncommon and it represents 23% of AION patients. In this study, AION was also common in age group between 40 to 50 years.

SUMMARY

This study was conducted to evaluate the causes of unilateral disc edema, age of presentation, sex preponderance, systemic associations, risk factors, treatment and prognosis. The results are as follows

- The most common cause of unilateral disc edema is anterior ischemic optic neuropathy [53.33%] and next common is optic neuritis [16.67%]
- Only type of AION presented to our clinic was NonArteritic type of AION.
- In the total cases of unilateral disc edema, 56.6% of cases were females and 43.3% were males. But males [75%] are commonly affected than females in NAION [25%]
- Mild grades of RAPD or a sluggish reacting pupil was seen with 93.8% of NAION where as high grades of RAPD was noticed with 60% of optic neuritis. Normal reacting pupil was seen in 66.7% of inflammatory disc edema
- 68.8% of NAION was associated with systemic illness, out of which 25% had DM, 12.5% had hypertension and 31.3% had both DM and HT.
- Uncontrolled hypertension and diabetes was noted in 50% of persons with DM and HT.

- MRI imaging was needed only in 40 % of the patients. It was very much useful in compressive neuropathy. MRI Brain in NAION showed small vessel ischemic changes in 6.67% of persons which indicates that the ischemic change were also noted in the brain.
- In NAION, disc showed sectoral pallor in 100% of patient, disc haemorrhages in 93.75% of patients. Fellow eye fundus of NAION patient showed small crowded disc [disc at risk] in 6.67% of patients.
- Inferior altitudinal defect was the most common field defect seen in NAION [31.25%] and the other type of field defect seen in NAION are inferionasal quadrantanopia [6.25%]and paracentral scotoma[6.25%].
- Associated diabetic retinopathy changes were seen in 6.67% of patient with NAION.
- Carotid Doppler study of 93.75% of patient with NAION showed a normal study, whereas only 6.25% of patient with NAION showed an insignificant stenosis in contralateral eye.
- Fellow eye involvement was seen in 12.5% of patients with NAION.
- 66.7% of the NAION patients showed disc pallor on follow up and all cases of optic neuritis showed temporal pallor on follow up.

- The visual prognosis of NAION is very poor even with prednisolone treated group [for patients without systemic illness or with controlled systemic factors] and with only control of systemic factors without oral prednisolone.
- All cases of optic neuritis showed improvement with intravenous steroids but it did not alter natural course of the disease.

CONCLUSION

If a patient with unilateral disc swelling presents to neuro-ophthalmology clinic, NA-AION [Non Arteritic Anterior Ischemic Optic Neuropathy] and ON [optic neuritis] should be considered first in the differential diagnosis. Differentiating NAION and ON is very essential as the treatment is entirely different for each condition. Other causes of disc edema should not be missed. A detailed history taking, visual field, color-vision and imaging tests [if needed, depending upon the cause of disc edema] should be performed for each and every case of unilatreal disc edema. Regular follow-up examination would be necessary for all cases to look for visual recovery and recurrence.

PART - III

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PROFORMA:

NAME:

AGE/ SEX:

I.P NO:

OCCUPATION:

CHIEF COMPLAINTS:

HISTORY:

PAST HISTORY:

PERSONAL HISTORY:

TREATMENT HISTORY:

GENERAL EXAMINATION:

BP:

OPHTHALMIC EXAMINATION:

	RE	**LE**
LIDS:		
CONJUNCTIVA:		
CORNEA:		
IRIS:		

ANTERIOR CHAMBER:

PUPIL:

Size

Shape

Light reflex

SWINGING FLASH LIGHT TEST: To detect RAPD

LENS:

ANTERIOR VITREOUS PHASE:

FUNDUS: (Direct and Indirect Ophthalmoscopy)

Disc (size, shape, margins)

Vessels

Macula

FUNDUS DIAGNOSIS:

VISUAL ACUITY:

Distant (without and with correction)

Near

INTRA OCULAR PRESSURE:

COLOUR VISION:

FIELDS:

PROVISIONAL DIAGNOSIS:

INVESTIGATION:

BLOOD SUGAR:

LIPID PROFILE:

TC:

DC:

ESR:

MANTOUX:

VDRL:

RADIOLOGICAL INVESTIGATION (IF NEEDED):

X-RAY CHEST

MRI BRAIN, ORBIT, SPINE

FINAL DIAGNOSIS:

TREATMENT:

Medical

Surgical

FOLLOW UP:

Every patient was asked for regular follow up after one week, 2 weeks, 4 weeks, 3 months and 6 months. At each visit visual acuity, colour vision, fields, slit lamp examination, fundus examination were recorded in all the patients.

INDEX TO MASTER CHART

1. Serial number:
2. Name:
3. Age: years
4. Sex: M-Male, F-Female
5. Visual Acuity: RE-Right Eye, LE-Left Eye, HM-hand movements, CFCF-counting fingers close to face, PL-perception of light.
6. Pupil right eye, pupil left eye, N-normal, IISUS-illsustained, RAPD-relative afferent pupillary defect.
7. Affected Eye: R-Right eye, L-Left eye
8. Field RE-right eye,LE-left eye, N-normal, INF ALT-inferior altitudinal defect
9. Colour vision- RE-right eye, LE-left eye, N-normal
10. Fundus media- CLR-clear, Hyperemia N-no, Y-yes, Disc Haemorrhage N-no, Y-yes

11. Other fundus findings-ART-arteriolar attenuation, G III HTN-grade III hypertensive retinopathy, NPDR-Non Proliferative Diabetic Retinopathy, VEN DIL-venous dilatation
12. Other eye fundus- ART ATT-Arteriolar Attenuation, VEN DIL-venous dilatation, NPDR-Non Proliferative Diabetic Retinopathy, GIII HTN-grade III Hypertensive Retinopathy.
13. DM-diabetes mellitus
14. HTN-hypertension
15. RETN- intraocular pressure in RE, LETN- intraocular pressure in LE
16. RBS- random blood sugar, SBP-systolic blood pressure, DBP-diastolic blood pressure, ESR-erythrocyte sedimentation rate
17. Mantoux test- ND-not done, N-normal
18. Carotid Doppler-N-normal, ND-not done,
19. Other investigations- AQ4-aquaporin 4 antibody, PT-prothrombin time, ND-not done

20. Imaging –ND-not done, MRI N-MRI normal, LETM-longitudinally extensive transverse myelitis, ON- optic neuritis.

21. Treatment-MP-methyl prednisolone, IS- immunosuppressive,AB-antibiotic

22. Diagnosis- AION-anterior ischemic optic neuritis.

MASTER CHART

SNO	NAME	AGE	SEX	REVN	LEVN	REPUPIL	LEPUPIL	AFFECTED EYE	RE FIELD	LE FIELD	RECOLORVN
1	VASANTHA	40	F	6/9	6/9	N	GI RAPD	L	N	IN QUAD ANOPIA	N
2	KALI	74	M	CFCF	NOPL	GI RAPD	N	R	NOT POSS	NOT POSS	NOT POSS
3	SELVAPANDI	50	M	6/24	6/6	GII RAPD	N	R	INF ALT DEF	N	N
4	SAMPATH	38	M	6/6	6/6	N	GII RAPD	L	INF ALT DEF	N	N
5	CHANDRA	48	F	HM	6/18	GI RAPD	N	R	NOT POSS	N	NOT POSS
6	ELIZABETH	61	F	6/18	CFCF	N	GI RAPD	L	N	NOT POSS	N
7	VENKATESAN	42	M	6/36	HM	N	GII RAPD	L	N	NOT POSS	N
8	ANAND	42	M	6/12	CFCF	N	GII RAPD	L	N	NOT POSS	N
9	ISMAIL MOIDEEN	54	M	6/24	6/12	SRL	N	R	INF ALT DEF	N	N
10	KATHIRVEL	34	M	6/36	0.5/60	GI RAPD	N	R	INF ALT DEF	NOT POSS	N
11	ABDUL RAHEEM	60	M	6/18	1/60	N	GII RAPD	L	N	NOT POSS	N
12	MUJBUR RAHMAN	46	M	6/9	6/6	GI RAPD	N	R	INF ALT DEF	N	N
13	SARAVANAN	46	M	6/60	6/18	GI RAPD	N	R	INF ALT DEF	N	DEF
14	SASIKALA	45	F	6/9	0.5/60	N	GII RAPD	L	N	NOT POSS	N
15	ALAGARSWAMY	55	M	0.5/60	6/12	GI RAPD	N	R	NOT POSS	N	NOT POSS

SNO	LECOLORVN	FUNDUS MEDIA	FUNDUS DISC MARGIN	HYPEREMIA	DISC HGE	SECTORAL PALLOR	OTHER FINDINGS	OTHER EYE FUNDUS	DM	HTN	RETN	LETN	RBS
1	N	CLR	BLURRED	Y	Y	Y	NO	N	NO	NO	12	12	80
2	NOT POSS	CLR	BLURRED	Y	Y	Y	NO	NO VIEW	NO	NO	10	10	89
3	N	CLR	BLURRED	Y	Y	Y	NO	DISC AT RISK	NO	NO	17.3	17.3	90
4	N	CLR	BLURRED	Y	Y	Y	VEN DIL	DISC AT RISK	NO	NO	13	13	103
5	N	CLR	BLURRED	Y	Y	Y	ROTH SPOTS	ROTH SPOTS	NO	NO	14.6	14.6	110
6	NOT POSS	CLR	BLURRED	Y	Y	Y	VEN DIL	N	NO	YES	17.3	17.3	104
7	NOT POSS	CLR	BLURRED	N	N	Y	ART ATTN	ART ATTN	NO	YES	14.6	14.6	110
8	NOT POSS	CLR	BLURRED	Y	Y	Y	ART ATTN	ART ATTN	YES	YES	18	16	152
9	N	CLR	BLURRED	Y	Y	Y	ART ATTN	ART ATTN	YES	YES	14.6	14.6	77
10	NOT POSS	CLR	BLURRED	Y	Y	Y	MOD NPDR	HIGH MYOPIC	YES	YES	12.6	14.6	475
11	NOT POSS	PCO	BLURRED	Y	Y	Y	ART ATTN	ART ATTN	YES	YES	12.6	12.6	225
12	N	CLR	BLURRED	Y	Y	Y	NPDR	NPDR	YES	NO	13	13	423
13	N	CLR	BLURRED	Y	Y	Y	ART ATTN	ART ATTN	YES	NO	12	12	120
14	NOT POSS	CLR	BLURRED	Y	Y	Y	ART ATTN	ART ATTN	YES	NO	12	12	104
15	N	CLR	BLURRED	Y	Y	Y	NPDR	NPDR	YES	NO	15	13	257

SNO	SBP	DBP	ESR	MAN TOUX	CAROTID DOPPLER	OTHER INV	CARDIAC	IMAGING/ VEP	TREATMENT	VN 1WEEK	VN 6TH MONTH FOLLOW UP	FOLLOW UP DISC PALLOR	RECUR RENCE	DIAG
1	110	80	15	N	N	ND	STABLE	ND	PREDNI	6/9	6/6	NO	NO	AION
2	120	80	9	N	N	ND	STABLE	ND	PREDNI	CFCF	CFCF	NO	NO	AION
3	110	70	13	N	N	ND	STABLE	ND	PREDNI	6/18	6/9	NO	NO	AION
4	110	70	7	N	N	PT - N INR - N	STABLE	MRI N	PREDNI	6/6	6/6	NO	NO	AION
5	120	80	110	N	N	ND	STABLE	ND	MP	HM	HM	YES	NO	AION
6	110	80	18	N	N	ND	STABLE	ND	NO	CFCF	CFCF	YES	NO	AION
7	220	140	15	N	N	ND	STABLE	MRI SVI	NO	CFCF	CFCF	YES	YES	AION
8	190	110	22	N	N	ND	STABLE	ND	NO	CFCF	CFCF	YES	NO	AION
9	140	90	12	N	N	ND	STABLE	MRI SVI	PREDNI	6/12	6/9	YES	YES	AION
10	120	80	12	N	N	ND	OLD MI	ND	NO	6/36	6/36	YES	NO	AION
11	110	80	12	N	N	ND	STABLE	ND	NO	1/60	1/60	YES	NO	AION
12	110	80	32	N	N	ND	STABLE	ND	NO	6/9	6/6	YES	NO	AION
13	120	80	12	N	N	ND	STABLE	ND	NO	6/60	6/12	YES	NO	AION
14	130	80	15	N	N	ND	STABLE	ND	NO	0.5/60	0.5/60	YES	NO	AION
15	110	80	5	N	N	ND	STABLE	ND	NO	0.5/60	0.5/60	YES	NO	AION

SNO	NAME	AGE	SEX	REVN	LEVN	REPUPIL	LEPUPIL	AFFECTED EYE	RE FIELD	LE FIELD	RECOLORVN
16	ALAGESAN	49	M	6/36	6/60	N	GI RAPD	L	N	PARACENT SCOT	N
17	AYISHA	30	F	NO PL	6/6	GIV RAPD	N	R	NOT POSS	N	NOT POSS
18	KALAIVANI	32	F	HM	6/6	GII RAPD	N	R	NOT POSS	N	NOT POSS
19	YUVARANI	22	F	6/6	PL	N	GIV RAPD	L	N	NOT POSS	N
20	REVATHY	23	F	6/9	NOPL	ILL SUS	GIV RAPD	L	N	NOT POSS	N
21	VELU	35	M	6/60	6/6	GI RAPD	N	R	PARACENT SCOT	N	DEF
22	SELVI	31	F	6/6	6/6	N	N	R	N	N	N
23	BHUVANESHWARI	24	F	CFCF	6/6	SRL	N	R	NOT POSS	N	NOT POSS
24	VELANKANNI	40	F	6/6	1/60	N	N	L	N	NOT POSS	N
25	PATTAMMAL	70	F	CFCF	6/18	N	N	R	NOT POSS	N	NOT POSS
26	JAYA	42	F	6/60	6/9	N	N	R	N	N	N
27	KAVITHA	46	F	6/6	1/60	N	N	L	N	NOT POSS	N
28	KARPAGAM	54	F	6/24	1/60	N	GIII RAPD	L	N	NOT POSS	N
29	RANI	46	F	6/6	6/18	N	GI RAPD	L	N	N	N
30	CHINNAPONNU	57	F	6/18	6/36	N	GI RAPD	L	N	N	N

SNO	LECOLORVN	FUNDUS MEDIA	FUNDUS DISC MARGIN	HYPERREMIA	DISCHGE	SECTORAL PALLOR	OTHER FINDINGS	OTHER EYE FUNDUS	DM	HTN	RETN	LETN	RBS
16	N	CLR	BLURRED	Y	Y	Y	GIII HTN	GIII HTN	YES	YES	12	12	140
17	N	CLR	BLURRED	Y	N	N	NO	N	NO	NO	14	16	80
18	N	CLR	BLURRED	Y	Y	N	NO	N	NO	NO	14	15	135
19	NOT POSS	CLR	BLURRED	Y	N	N	NO	N	NO	NO	12.2	12.2	106
20	NOT POSS	CLR	BLURRED	Y	N	N	VITRITIS	PALLOR	NO	NO	10	12	131
21	N	CLR	BLURRED	Y	N	N	NO	N	NO	NO	16	13	134
22	N	VITRITIS I	BLURRED	Y	N	N	TOXOPLASMOSIS	N	NO	NO	10.2	10.2	80
23	N	VITRITIS I	BLURRED	Y	N	N	CHOROIDITIS MACULA	N	NO	NO	10.2	10.2	110
24	NOT POSS	VITRITIS I	BLURRED	Y	N	N	PERIPHLEBITIS	N	NO	NO	15	12	110
25	N	VITRITIS I	BLURRED	Y	Y	N	HARD EXUDATES MACULA	N	YES	YES	12.2	12.2	152
26	N	CLR	BLURRED	Y	N	N	MACULAR STAR	N	NO	NO	8.5	10.2	78
27	NOT POSS	CLR	BLURRED	Y	N	N	MACULAR STAR	N	YES	YES	10.2	10.2	102
28	NOT POSS	CLR	BLURRED	Y	N	N	CHOROIDAL FOLDS	N	NO	NO	10	14	78
29	DEFECTIVE	CLR	BLURRED	Y	N	N	CHOROIDAL FOLDS	N	YES	YES	17.3	17.3	100
30	DEFECTIVE	CLR	BLURRED	Y	N	N	CHOROIDAL FOLDS	N	NO	NO	14.6	14.6	105

SNO	SBP	DBP	ESR	MANTOUX	CAROTID DOPPLER	OTHER INV	CARDIAC	IMAGING/ VEP	TREATMENT	VN 1WK	VN 6TH MONTH FOLLOW UP	FOLLOW UP DISC PALLOR	RECURRENCE	DIAG
16	140	90	15	N	R NON SIG STENOSIS	ND	STABLE	ND	PREDNI	6/60	6/36	YES	NO	AION
17	130	80	15	N	ND	AQ4 POS	ND	MRI LETM	MP + IS	0.5/60	6/60	YES	NO	OPTIC NEURITIS
18	110	80	7	N	ND	ND	STABLE	MRI ON	MP	6/60	6/6	YES	NO	OPTIC NEURITIS
19	110	70	11	N	ND	ND	ND	MRI ON	MP	6/24	6/6	YES	NO	OPTIC NEURITIS
20	120	70	4	N	ND	ND	ND	MRI N	MP	6/60	6/6	YES	YES	OPTIC NEURITIS
21	120	80	12	N	ND	ND	ND	MRI ON	PREDNI	6/60	6/36	YES	NO	OPTIC NEURITIS
22	120	80	3	N	ND	TORCH +	ND	ND	AB + STEROID	6/6	6/6	NO	NO	POST UVEITIS
23	110	80	10	POS	ND	TORCH +	ND	ND	AB + STEROID	6/60	6/6	NO	NO	POST UVEITIS
24	120	80	12	N	ND	ND	ND	MRI SCLERITIS	AB + STEROID	6/60	6/60	NO	NO	PANUVEITIS
25	110	80	13	POS	ND	ND	ND	ND	AB + STEROID	2/60	6/18	NO	NO	NEURORETINITIS
26	120	80	9	N	ND	ND	ND	ND	AB + STEROID	6/36	6/6	NO	NO	NEURORETINITIS
27	110	70	15	N	ND	ND	ND	ND	AB+STEROID	6/60	6/12	NO	NO	NEURORETINITIS
28	120	80	9	ND	ND	ND	ND	MRI ETH MOIDAL MUCO CELE	DECOMP RESSION	CFCF	NO PL	YES	NO	COMPRESSIVE OPTIC NEUROPATHY
29	100	70	15	ND	ND	ND	ND	MRI INTRA CONAL HAEM ANGIOMA	ORBITOTOMY MASS REMOVAL	6/12	6/12	NO	NO	COMPRESSIVE OPTICNEUROPATHY
30	130	80	25	ND	ND	ND	ND	MRI EXTRA CONALMASS	ORBITOTOMY MASS REMOVAL	6/36	6/12	NO	NO	COMPRESSIVE NEUROPATHY